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CARCINOMA ARISING FROM CHRONIC GASTRIC ULCER[1]

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IN the more recent medical literature much attention has been directed toward the problem of gastric carcinoma arising from chronic peptic ulcer. Pathologists and surgeons are equally interested as both the problem of etiological relationship between chronic stimulus and tumor and the question of operative treatment of chronic gastric ulcer are involved. As a pathological change is subject to malignant degeneration, more radical treatment, of course, is required than for a lesion devoid of this menace.

The theory that malignant degeneration of chronic peptic ulcer is possible cannot be denied. There is ample evidence that malignant tumors may start in the edge of a chronic ulcer, for example kangri cancer, lupus cancer, carcinoma arising in pressure ulcers of the tongue, etc. That long continued stimuli may produce cancer of the stomach has been demonstrated by J. Fibiger. In cancers formed in a gastric ulcer such a stimulus may be represented by the niche where food particles are constantly undergoing decomposition; Mayo found malignant change three times in 42 cases of diverticulum of the large bowel.

The actual relationship between peptic ulcer and cancer, however, is another question. Is there a definite relation between them, and if there is how close is this relationship? What are the chances of a chronic peptic ulcer becoming malignant? Much has been written on the subject and the opinions brought forward differ greatly. Rokitansky was one of the first to allude to the possibility of the incidence of peptic ulcer and cancer of the stomach. In 1878, Lebert reported 8 cases of gastric cancer in which typical symptoms of peptic ulcer were observed for many years before death. Hauser was the first to furnish histological evidence of cancer arising from the edge of a chronic ulcer. Since then many reports have been made of such cancers and some of these reports contain data as to the frequency of such lesions. Unfortunately, not all of these statistics can be used for comparison as many of the reports obviously are not complete. In only a few instances is the percentage given of gastric ulcers becoming malignant and of carcinoma probably arising from chronic ulcer. Most of the statistics published mention but one of these items, the "frequency of ulcer cancers."

As stated the opinions of different authors vary greatly. According to A. Nielsen there is not sufficient evidence to prove that cancers develop on a chronic ulcer base. Hirschfeld does not accept the theory of an etiological relationship between ulcer and cancer. In the opinion of R. Schmidt, patients suffering from chronic ulcer are immune to carcinoma. Henke and Stromeyer stress the rarity of cancers arising from gastric ulcers. Walton expresses the opinion that such cancers are more common than generally believed. According to Fuetterer a great many pyloric cancers arise from chronic ulcers. Table I shows a few statistics on malignant degenera-

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tion and discloses the discrepancies in opinions. If we accept only those compilations which furnish clear and complete figures and reject all others, the reasons for the differences in opinion may be discussed under three heads: (1) the specimen itself; (2) the lack of uniformity in technique in studying the specimen; and (3) the lack of criteria on which to base a diagnosis.

1. *The specimen.* The pathologist is more likely to see fewer cases of cancer arising from gastric ulcer than the surgeon who has the opportunity of resecting early cases. When a case comes to autopsy the tumor has progressed so far that the entire floor of the ulcer is so extensively destroyed that it is almost impossible to determine whether or not there had been a pre-existing ulcer (Payr).

TABLE I.—STATISTICS AS TO MALIGNANT DEGENERATION

Author	Carcinomatous degeneration of ulcer per cent	Carcinomata arising from chronic peptic ulcer per cent
Borrmann		1
Paterson	1	None
Garré	3 to 5	
Orator	8.5	10 to 15
Payr	26	
Moynihan	18.5	about 66
Newcomb	3.5	13
McCarty and Wilson		71
McCarty	68	
Brinkmann	5.5	

2. *Technique of studying specimen.* Statistics based upon clinical and macroscopical observations only should be discarded for, as will be pointed out later, positive proof of cancer developing on an ulcer base can be had only upon microscopic examination. Even material worked up with the utmost care may show marked deviations, depending on what cases are included in the series. It should be remembered that many surgeons are very radical in that they resect almost all ulcers, even subacute ones, coming to their care. If all ulcers observed are included, the rate of carcinomatous degeneration would be very low. In our opinion all ulcers should not be included. Acute or even subchronic ulcers are not considered as liable to malignant change. Therefore, only callous ulcers, positively chronic, should be included. If only these are accepted, the percentage will be much higher. Most of the articles published are not clear on the point as to whether or not the ulcer is of the long standing callous type. It is for this reason, we believe, that the statistics show such marked discrepancies.

3. *Criteria for diagnosis.* Opinion is not yet uniform as to the criteria for, and possibility of, determining that a cancer has developed from an ulcer. We should like to go more fully into detail as to this question before commenting upon our own cases.

The symptoms of cancer secondary to ulcer will be dealt with from three standpoints: (1) the clinical; (2) the pathological and macroscopic; and (3) the histological features.

From a clinical standpoint, the symptoms point to the possibility of an ulcer undergoing cancerous degeneration if the symptoms of ulcer—pain after taking food, pyrosis, sour eructation, etc.—after long standing, years or decades, gradually change into typical symptoms of gastric cancer—almost permanent dull ache, aversion to food, rapid loss of weight, cachexia. Of course, even more convincing are characteristic changes in the laboratory and X-ray findings. In several cases in which gastro-enterostomy had been performed many years previously, malignant degeneration of gastric ulcer was observed at a second operation. We had a case in our clinic in which there was a 5 year interval between the operations. Unfortunately, it was possible to examine only a lymph gland near the pylorus in which adenocarcinoma was demonstrated.

All cases of gastric cancer with normal or even increased hydrochloric acid should arouse suspicion of malignant degeneration of peptic ulcer. Moreover, long anamnesis in itself points to previous ulcer, as according to Lebert 83 per cent of patients with gastric cancer die within 18 months, 9 per cent within 2 years, the remaining 8 per cent within 4 years, the latter being without exception scirrhous cancers. Such cases as that of Paterson—one patient alive 3 years, the other alive 4½ years after gastro-enterostomy for inoperable cancer—are extremely rare.

These symptoms, however, do not give entirely satisfactory data for the diagnosis of cancer secondary to ulcer. Often laparotomy

Fig. 1. Chronic peptic ulcer showing unchanged relation of muscularis mucosæ and muscular coat. Iron hæmatoxylin stain. ×1.5.

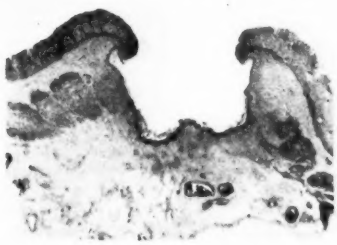

Fig. 2. Progressive separation of muscularis mucosæ and muscular coat toward the ulcer edge in chronic peptic ulcer. Iron hæmatoxylin stain. ×1.5.

in a case which seems typical for ulcer, reveals no ulcer. In such cases, according to Hauser and Versé, the underlying pathological change is chronic gastritis. On the other hand, absence of symptoms does not exclude the possibility that ulcer has been present as has been demonstrated by the accidental finding at autopsy of an ulcer and the great number of sudden perforations without a previous history of ulcer. As to the malignant degeneration of an ulcer for which a gastro-enterostomy has been performed, which malignancy is detected at a second operation, it is questionable whether the ulcer was not already malignant at the time of the first operation. If considerable time has elapsed between the first and second operations, the probability of the latter assumption decreases, especially if the tumor is of small size at the time of the second operation.

Macroscopic examination of the operative specimen gives more certain findings. In the first place the location of the lesion is of importance. Malignant degeneration of a duodenal ulcer is extremely rare. On the other hand, malignant change in a pyloric or prepyloric ulcer is found ten to fifteen times more often than in ulcers situated higher along the lesser curvature. Ulcers 2 centimeters or more in diameter are said to be especially liable to malignant degeneration.

It is rather difficult to give a comprehensive macroscopic picture of a cancer secondary to ulcer, as the picture varies according to the stage of development. In the beginning, sometimes even in a rather advanced stage, it cannot be distinguished from a simple, chronic, peptic ulcer, all the characteristic features of which it possesses. In some cases there is a sharply circumscribed round or oval defect in the mucosa, at the edges of which the mucosa is rolled inward and fixed to its base. In other cases the edges are undermined, overhanging, and rigid. The mucosa around the ulcer is often thrown into radial folds. The floor of the ulcer consists of dense scar tissue, smooth and never crumbling; in extreme cases it may be 2 centimeters thick. The defect often has the shape of an inclined funnel, the steep wall of which is at the cardiac edge, while it slopes gently toward the pyloric edge. This shape is very probably characteristic of chronic peptic ulcer, as, according to most authors, it corresponds to the area containing the normal arterial supply of the stomach. Primary cancer destroys all vessels with which it comes in contact and a shape characteristic for the obstruction of only one of the larger vessels is an exception and will develop only by chance.

The most convincing signs of peptic ulcer are observed in cross sections of the ulcer. We find there the following characteristic picture of the muscular coat: At the outer edge the muscle fibers radiate as a sharply limited, tight strand, in an acute or even a right angle, to the ulcer floor where they are lost. This condition is the result of a cicatricial contraction by which the musculature is pulled toward the center of the ulcer where it will eventually be entirely destroyed. No muscle tissue is found in the ulcer base. If a pre-existing cancer becomes ulcerated, this behavior of the muscle coat is never observed, as the muscle becomes "grubbed up" and the fibers are widely spread by the invading

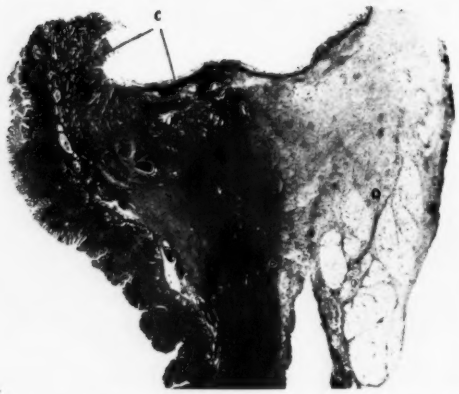

Fig. 3. Case 1. Photomicrograph showing the edge of the ulcer. *m*, Muscular coat; *c*, carcinomatous glands in ulcer base. ×3.8.

neoplasm. Another characteristic sign, sometimes visible with a hand lens but more often seen only with the microscope, is fusion of the muscularis mucosæ and the muscular coat at the edge of the ulcer. The importance of this sign will be dealt with in the histological discussion.

Very often extensive peritoneal adhesions are present. The omentum may be fixed in radial folds to the base of the ulcer, and sometimes the base of the ulcer is formed merely by these adhesions. In cancer, extensive adhesions are seldom present, even in case of complete perforation.

If the ulcer shows only the signs described, it cannot be distinguished from a simple chronic ulcer. At times, however, at some part of the ulcer edge, a medullary swelling of the mucosa is observed, which may extend into the ulcer base. The cut surface shows a white, soft nodule. Care should be taken in any case as the prepyloric mucosa is prone to produce benign hyperplasia and the picture may be similar. Diagnosis will depend on microscopic examination. Later the tumor invades the ulcer base and its environs, and after the breaking down of the neoplastic tissue the picture of the original callous ulcer will be entirely blurred. In this stage a typical, crater-like ulcer is seen, with irregular, everted hard edge and an uneven, crumbling floor; or in cases of scirrhous cancer, there is a widespread callous thickening of the stomach wall. In this phase the ulcer cannot be distinguished from a primary cancer.

I will mention here a very rare and interesting form of cancer secondary to ulcer—a scar cancer. In several instances there was observed in the linear scar of an hour-glass stomach, a circumscribed medullary tumefaction, which, on microscopic examination proved to be a carcinoma.

Summarizing the principles of macroscopic diagnosis of cancer secondary to peptic ulcer those signs are to be taken into consideration which show that scarring took place in the stomach wall previously not invaded by tumor and medullary swelling of the mucosa at the edge of the ulcer. The most valuable signs are the typical form of the ulcer defect, that is the turning up of the muscle layer toward the ulcer floor and external adhesions.

As shown, macroscopic diagnosis is not always dependable, whereas histological examination will establish the diagnosis with certainty in most cases, provided proper care has been used in making the examination. Two problems arise in making the histological examination: (1) the seeking of evidence to determine the presence of cancer and (2) the seeking of evidence that the cancer started in the margin of a pre-existing callous ulcer.

The first problem cannot always be easily solved. As mentioned, the prepyloric mucosa is prone to produce hyperplastic growth. Glands of this hyperplastic mucosa are sometimes not entirely regular, their cells may show some polymorphism and be stained darker than those of the surrounding mucosa—the much debated "dark cells" of the literature. It is at times difficult to distinguish the growth from adenocarcinoma. In determining malignancy personal feeling is liable to play a great rôle, as unmistakable criteria are often lacking. This fact may explain in part the wide range of malignant degeneration in chronic ulcers as described by different authors. In our opinion it should be assumed that cancer is present only if, in addition to the known symptoms of malignancy, there can be no mistake in interpreting other signs, for instance the presence of a distinct neoplastic infiltration of the submucosa. A

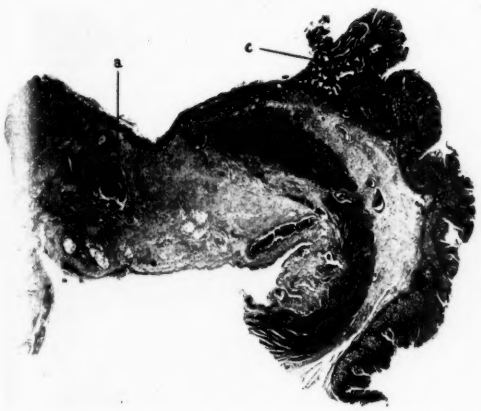

Fig. 4. Case 2. Edge of ulcer. *c*, Carcinoma; *a*, endarteritic vessel. ×3.3.

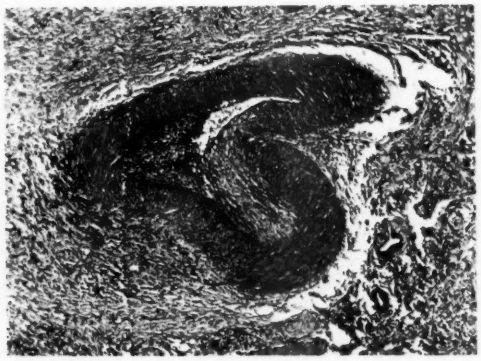

Fig. 5. Case 2. Vessel shown in Figure 4. ×30.

few isolated glands do not necessarily indicate malignant change.

It should be emphasized that it is not enough to examine one block of tissue taken from the edge of an ulcer. Although in many cases the cancer will have invaded the entire circumference and thus can be demonstrated in any block taken at will, there are a great many cases in which the tumor is confined to a very small part of the ulcer margin. Preparing serial sections from the entire ulcer area in every case is not feasible; 4 to 6 blocks at least, must be examined, however.

As to the second question, the search for microscopic signs of callous ulcer, it will be noted: (1) The ulcer floor consists of a mass of granulation and scar tissue which becomes more fibrous toward the deeper layers. This tissue occupies the entire ulcer base and spreads beyond the margins of the ulcer as a cicatrized broadening of the submucosa. It may reach as deep as the serous coat and in the presence of external adhesions it may involve the organ to which it adheres—the omentum, the pancreas, etc. (2) The ulcer base is entirely devoid of muscle tissue. At the ulcer edge the muscular coat radiates as a sharply demarcated, tight strand, in an acute angle, into the floor of the ulcer. The muscularis mucosæ is very often pulled, in the process of scar tissue formation, to the muscular coat. Thus both layers become closely approximated or even fused at the ulcer margin. Newcomb found this condition, at least in some part of the ulcer margin, in 98.8 per cent of his cases. In the study of our material we were not able to confirm this point as in 64 cases of chronic peptic ulcer we found actual fusion in but 34 cases, some approximation in 22 cases, and unchanged position (Fig. 1) in 5 cases. In 3 cases distinct progressive separation of both layers toward the ulcer edge (Fig. 2) could be observed. Undoubtedly, if scarring should proceed without disturbance for a considerable length of time, the muscular layers would become approximated. It is known, however, that the natural history of a peptic ulcer includes periods of ulceration alternating with periods of healing. Whether or not the sign described will be found depends largely on the phase which happens to prevail at the time of examination. That scarring does not necessarily cause fusion of the muscle layers is shown also by the fact that in most cases of even entirely quiescent ulcers, scarring is not noticeable completely around the ulcer edge but only in some parts of it. (3) Obliterated, endarteritic vessels can be found in the base of the vast majority of peptic ulcers. Many pathologists believe that they are of etiological importance. Endarteritis is also observed in primary cancers, but occluded vessels of larger caliber, 1 to 2 millimeters in diameter, deep in the ulcer base, surrounded by tissues showing only a moderate or no inflammatory reaction, occur almost exclusively in chronic peptic ulcers.

Fig. 6. Case 3. Operative specimen. ×0.5.

If somewhere in the margin of an ulcer presenting the criteria mentioned, carcinomatous proliferation is noted, and at the same time the greater part of the ulcer margin and base are tumor free, there can be no doubt as to the diagnosis—cancer arising from a chronic peptic ulcer. Cancer detected in this stage is comparatively rare. In most cases cancer tissue is found completely around the periphery of the ulcer. This phenomenon may be the result of two things: (1) the neoplastic process may have started simultaneously at several points in the ulcer margin, the stimulus being present in all parts of the ulcer edge, or (2) as the tumor could not penetrate into the dense, fibrous ulcer base, the growth took place around the margins which offer less resistance. The tumor cells may often be absent in the entire thickness of most of the ulcer base. In other cases the dense, cicatrized layers extending toward the gastric lumen contain no tumor cells, but the subserous layers are infiltrated. This is explained by the fact that it is easier to invade the loose tissues. Stromeyer does not recognize lesions in this not entirely initial stage as cancers arising from ulcers but considers them as primary cancers ulcerated away and cicatrized to such an extent that no cancer cells can be observed in the scar tissue. Moreover, he suggests that the rate of scar tissue formation may exceed that of the neoplastic growth and in this way the entire ulcer base may become cancer free. According to him diagnosis in these cases should not be *carcinoma ex ulcere* but *ulcus in carcinomate*. Borrmann is of the same opinion and believes that cancers from ulcers are rare. He accepts only those cases in which but a small part of the ulcer margin is occupied by tumor and in which the ulcer base is entirely devoid of cancer cells. He does not believe in a close etiological relationship between ulcer and cancer, as he found malignant change in only 1 per cent of all ulcers. He puts forth the following considerations: (1) How can it be explained that in the superior horizontal part of the duodenum where ulcer is so frequent, cancer is a rarity, whereas in the region of the ampulla where ulcer is exceptional, cancer is rather often seen? (2) It is difficult to realize how a cancer produces an annular growth around the ulcer margin, as cancer in general shows a uniform centrifugal growth. Until it reaches the opposite margin after encircling the ulcer border, extensive infiltration far in all other directions must have resulted. In this stage a large carcinomatous infiltration with eccentric ulcer is to be expected rather than a lesion with a uniform annular border. That the neoplasm begins simultaneously in several points, which in some cases seems to be more than probable, Borrmann cannot believe. He mentions the queer fact that no cases are known in which the greatest part of the ulcer edge is occupied by cancer but only those cases are mentioned in which the tumor is limited either to a small focus or in which the tumor forms a closed ring. Since Borrmann's contribution, Klein and Demuth have reported the first case of cancer secondary to ulcer in which the neoplastic ring was not entirely closed.

It is true that very few cases recorded in the literature give exact data as to the extension of the malignant involvement. We believe that as more cases are examined thoroughly there will be an increase in the number in which the neoplastic ring is nearly closed. As far as we know, Case 4 in our series is the second case reported in the literature. Klein and Demuth advance the following arguments against the theory of Stromeyer: Ulceration of the carcinomatous tissue may be acute or chronic. In the acute type signs of acute reaction and masses of necrotic tumor tissue must be present in the ulcer base, which must be infiltrated even in the chronic case, as according to Stromeyer cancer is already present

when the ulceration and scarring commences, at which time there is no rigid scar tissue but only succulent, cellular granulation, which is easily infiltrated by carcinoma. Complete sloughing away of the tumor by a phlegmon or abscess, as suggested by Stromeyer, is highly improbable, as in the ulcer base entirely or nearly normal vessels of considerable size, evidently not newly formed ones, are always present. In extensive pyogenic destruction, through which the entire tumor would slough away *en masse*, leaving not a trace behind, the vessels must have suffered considerable damage. Wasting away of the tumor, through pressure of its own stroma, can be ruled out, as cancers secondary to ulcers are almost without exception highly cellular adenocarcinomata entirely lacking in scirrhous features. The opinions of Stromeyer and Borrmann are not accepted by Hauser who says that it is difficult to realize that exactly in the center of a carcinoma a peptic ulcer would develop to destroy evenly almost the entire tumor and to leave but a narrow margin. According to the great majority of contributors, those callous carcinomatous ulcers the greater part of the floor of which is free from cancer can be safely regarded as cancers secondary to ulcers.

Of course, in time the tumor will invade the entire ulcer base. Considerable difficulty may be met with in diagnosing these cases. Often, if the ulcer base is not too extensively destroyed, fair evidence of previous peptic ulceration may be present. Here too the symptoms and signs of chronic ulcer must be searched for. Later in the process of the disease the original structure may be unrecognizably blurred by the neoplastic growth. In such cases, of course, it would be impossible to state that the cancer had arisen on an ulcer base. Very probably in this stage of the lesion many cases are seen and the origin of the lesion is not even suspected.

We shall mention another diagnostic sign stressed by several authors but in our series found to be entirely without dependence, namely, the different locations of concomitant gastric lesions. It is alleged that with peptic ulcer and secondary cancer pyloric gastritis prevails, whereas cancer is generally associated with diffuse gastritis.

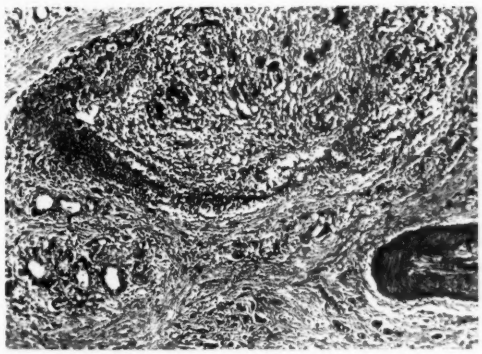

Fig. 7. Case 3. Tubercle and carcinoma in ulcer base. ×75.

In the relation between ulcer and cancer, another possibility should not be overlooked, namely, that a carcinoma arising independently in the vicinity of the ulcer would break into the latter. In this case, however, the tumor tissue is most voluminous at its starting point and not at the ulcer margin.

AUTHOR'S CASES

Our material consists of operative specimens removed at the Third Surgical Clinic of the University of Budapest from September 1, 1929, to January 1, 1933.

Number of specimens	166
Gastric ulcer	
Chronic	64
Subchronic and subacute	7
Duodenal ulcer	41
Jejunal ulcer	4
Gastric carcinoma	28
Linitis type	2
Gastric lymphosarcoma	1
Hypertrophy of pylorus	2
Duodenal diverticulum	1
Gastritis	1
Tuberculosis of stomach	1
Palliative resections for duodenal ulcer	8
Cancers secondary to ulcer	6

In every case of cancer secondary to ulcer at least 4 blocks from different parts of the ulcer edge have been removed for histological examination, except in Case 1, which unfortunately was not worked up systematically and therefore we are unable to give the topography of the tumor.

CASE 1. A man, aged 57 years, came to the clinic for severe hunger pains, of 1 year's duration, sour eructations, and hæmatemesis. In spite of a good

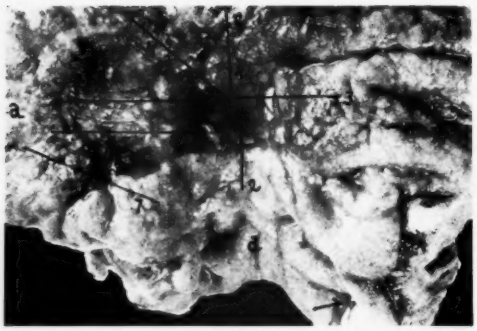

Fig. 8. Case 4. Part of operative specimen. *c*, Cardiac edge; *d*, duodenal edge; *a*, anterior wall; *p*, posterior wall. Arrow indicates entrance of diverticulum. Natural size.

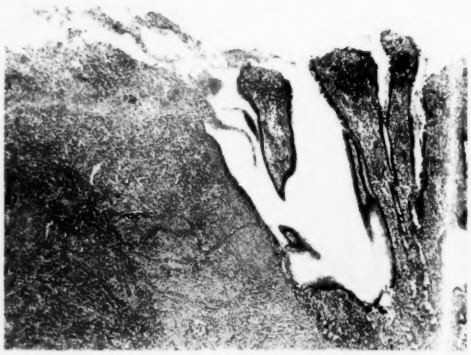

Fig. 9. Case 4. Anterior stomach wall edge, block 3. Benign papillary hypertrophy at ulcer edge. ×27.

appetite he lost 6 kilograms in weight. Test meal yielded free hydrochloric acid, 40; total acidity, 60. At laparotomy a stellate scar was found in the distal third of the lesser curvature, to which the gastrocolic ligament and the greater omentum were adherent. The hepatogastric ligament was shortened and inflamed. Extensive resection of the stomach was performed. In the specimen removed a round prepyloric ulcer was found, 45 millimeters in diameter. It had the shape of a shallow funnel and had a smooth, firm, whitish floor and elevated, strongly overhanging edges. Its base was formed by a thin layer of scar tissue and adherent omentum. The cut surface showed a pea sized, white, soft nodule just beneath the edge of the mucosa. This nodule was present in three sections taken from different parts of the ulcer.

Histology (*Fig. 3*). The ulcer base was formed by granulation tissue becoming highly fibrous toward the depth. The surface was covered with a thin layer of necrotic material. At the center the fibrous layer was very thin and underneath it the base of the ulcer was formed by the fatty tissue of the omentum. The muscular layer radiated under a right angle into the ulcer floor, where it was lost. The space between the muscularis mucosæ and the muscular coat was rather increased by scar tissue broadening of the submucosa, which could be followed for about 13 millimeters from the ulcer edge. At the ulcer edge both layers became even more separated by invasion of a highly atypical glandular tissue. In one of the blocks a gradual transition from normal gastric glands into carcinomatous ones could be observed. In the other blocks too, the ulcer edge was formed by adenocarcinoma, no transitions, however, being observed. The neoplastic tissue seemed to invade the mucosa rather from below. Groups of atypical glands decreasing in size toward the center were seen for about 12 millimeters from the ulcer margin. In the center of the ulcer base an area of about 20 millimeters in diameter was entirely free from neoplasm. In the fibrous tissue, several arteries of 0.25 to 0.5 millimeters in diameter containing organized thrombi were seen. Cells of the neoplastic glands showed considerable polymorphism and a great number of mitotic figures.

Case 2. A man, aged 66 years, complained of epigastric pain and vomiting of 1 year's standing. In the last months he vomited coffee-grounds-like masses. In the epigastrium a tender mass could be palpated. Because of the bleeding no test meal examination was carried out. At operation a mobile tumor the size of a child's fist was found near the pylorus on the lesser curvature. The distal two-thirds of the stomach was resected. In the specimen a prepyloric, funnel shaped ulcer with slightly raised and overhanging borders, about 40 millimeters in diameter, was seen. The ulcer floor consisted of a hard, smooth, whitish tissue. In the cross section of the ulcer an upward turning of the muscular coat was conspicuous. At the cardiac edge a pea sized, white nodule was noticed beneath the mucosa. Six blocks were removed for microscopical examination.

Histology (*Fig. 4*). The ulcer base was formed by the granulation tissue becoming highly fibrous toward the depth and was covered by a necrotic layer. In the connective tissue, which presented heavy lymphocytic and eosinophilic infiltration, several large endarteritic, partially obliterated arteries were seen (Fig. 5). The scar tissue broadening of the submucosa could be followed far from the ulcer margin beneath the mucosa. At the ulcer edge the muscular coat radiated as a compact strand into the ulcer floor, at an angle of about 60 degrees, and then was lost. At some points a close approximation of the muscularis mucosæ and the muscular coat could be observed. All around the margin of the ulcer the mucosa presented a similar picture. Near the margin of the ulcer the glands became deeper, were ramified, and showed lumina filled with papillary projections. The glands penetrated the muscularis mucosæ and the superficial layer of the muscular coat; at some places having reached even the subserous tissue.

Fig. 10. Case 4. Duodenal edge, block 2. Rarefied area shown by arrow. *c*, "Swarm" of epithelial cells invading submucosa. ×27.

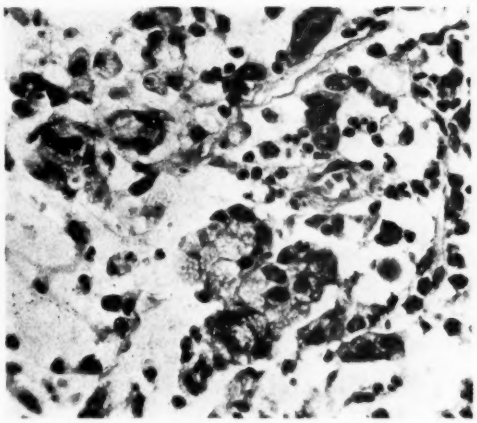

Fig. 11. Case 4. High power photomicrograph of rarefied area of Figure 10. ×425.

The glands were lined with several layers of tall cylindric epithelium which showed moderate polymorphism and a few mitotic figures. In general, atypical glands were found only a few millimeters from the edge of the mucosa; at some points, however, gland groups were seen in the base of the ulcer, 10 to 12 millimeters from the mucosa. Most of the ulcer base was tumor free.

Case 3. A woman, aged 57 years, complained of pains and cramps in the gastric region, especially severe after meals. During her illness of 2 years, she had lost 24 kilograms. Test meal revealed free hydrochloric acid, 32; total acidity, 62. At laparotomy in the prepyloric region, a tumor the size of a child's fist, freely movable, was detected. Along the lesser curvature there were several firm lymph glands. Resection of the stomach was performed. In the operative specimen on the lesser curvature, just next to the pylorus there was a round, shallow, terraced ulcer (Fig. 6), about 35 millimeters in diameter, with rolled in edges. The ulcer floor consisted of smooth, firm, whitish tissue. Cross section showed upward turning of the muscular coat. Six different blocks have been examined.

Histology. The ulcer consisted of a highly fibrous, scar tissue, heavily infiltrated with lymphocytes, eosinophils, and plasma cells covered with a thin, superficial layer of fibrinoid material. The muscular coat radiated at the ulcer edge as a compact strand into the ulcer floor. At the cardiac margin the muscularis mucosæ and the muscular coat were fused. At the pyloric edge, the glands suddenly became very irregular, and they invaded the deeper layers, having infiltrated also the submucosa as much as 10 to 15 millimeters beneath the normal mucosa. In all other blocks, which presented essentially the same picture, no transition could be observed; moreover, it was clearly discerned that the tumor invaded from below upward. Groups of irregular glands rapidly decreasing in size and number toward the center were seen throughout the entire ulcer base. Under low power magnification, there were, however, entire fields free from cancer. In some sections in the depth of the ulcer base tubercle-like formations were seen with many epithelioid and giant cells (Fig. 7). From the periphery they were being invaded by tumor tissue. No foreign body was seen within these nodules nor could acid fast bacilli be demonstrated.

Case 4. A woman, aged 62 years, complained of gastric distress after meals. This condition had been present for 20 years. For the past 2 years her condition had grown progressively worse. Within 7 months she had lost 12 kilograms in weight. Test meal yielded free hydrochloric acid, 34; total acidity, 49. X-ray examination revealed a duodenal diverticulum and a rigid prepyloric area. At laparotomy the prepyloric area was found rigid and bulky. There

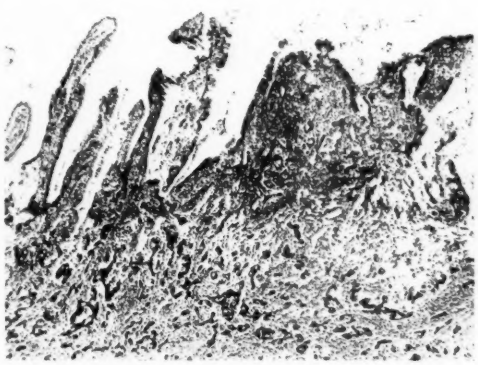

Fig. 12. Case 4. Frank adenocarcinoma at duodenal edge. ×26.

Fig. 13. Case 5. Early carcinoma near ulcer edge. ×20.

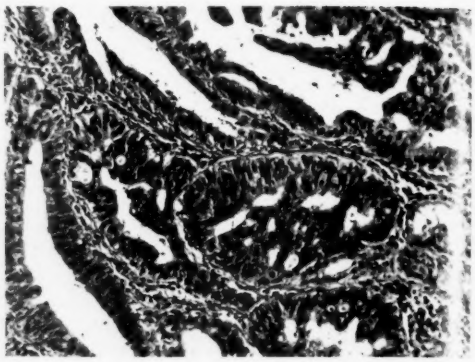

Fig. 14. Case 5. The part outlined in Figure 13. ×1[illegible].

were extensive adhesions to the mesocolon and the head of the pancreas. Resection of the stomach was done.

In the specimen removed (Fig. 8), a duodenal diverticulum was seen (arrow points to its entrance). At the lesser curvature, 3 centimeters from the pylorus, there was a hemispherically depressed ulcer defect the size of a half walnut. Its floor and walls were smooth, firm, whitish. Joining it on the anterior stomach wall was a semicircular, more superficial defect of the mucosa, with hyperæmic borders and a moderately firm, freshly digested floor. No enlarged lymph glands were present. In cross section, upward turning of the muscular coat was observed. Seven blocks were removed for microscopic examination, as shown in Figure 8: *1*, cardiac; *2*, duodenal; *3*, posterior; *4* to *7*, anterior stomach wall edge.

Histology. Block *3* presents a typical picture of chronic peptic ulcer with marked papillary hyperplasia of the mucosa (Fig. 9). In the highly fibrous ulcer base there were several vessels showing endarteritis. The muscular coat radiated into the ulcer floor. The muscularis mucosæ and the muscular coat were closely approximated, almost fused. At first glance blocks *1* and *2* presented a similar picture except for an absence of papillary hyperplasia. At closer inspection, however, a peculiar change was seen within the mucosa (Fig. 10). In some places just at the ulcer margin, at other places somewhat farther from the margin quite small areas, 1 to 2 millimeters in diameter, of decreased density were observed, showing loss of normal structure of the mucosa. Under high power magnification in an almost homogenous mass unstained both by hæmatoxylin and eosin, but showing pink metachromasia with thionine stain, fragments of glands and scattered small groups of epithelial cells besides some round cell infiltration were seen (Fig. 11). Many of the scattered epithelial cells were vacuolar or actual signet ring cells, some of them had bursted and blended into the homogenous mass mentioned. At the duodenal edge a narrow mass of free epithelial cells was seen advancing between the mucosa and the muscularis mucosæ. Blocks *4*, *5*, *6*, and *7* showed acute digestion of the superficial layers and a marked papillary hypertrophy of the mucosa. In the deeper layers typical gland groups of mucoid adenocarcinoma were present (Fig. 12), not farther, however, than about 3 to 4 millimeters from the ulcer margin. The entire ulcer base was tumor free.

As mentioned, as far as we know, this is the second known case of the nearly closed ring type of cancer secondary to ulcer.

Case 5. A man, aged 44 years, had typical ulcer anamnesis for 3 years. In the last year the pains had become almost constant and he had lost 23 kilograms in weight. Test meal yielded free hydrochloric acid, 70; total acidity, 100. X-ray examination showed the typical ulcer deformity of the bulbus duodeni. At laparotomy a perforating ulcer was found on the posterior stomach wall just anterior to the pylorus. It was freed from the pancreas with considerable difficulty, the center of the ulcer base being left on the pancreas and cauterized. Then resection of the stomach was performed.

The specimen removed showed a typical callous ulcer, 30 millimeters in diameter. In the center of its base there was a hole, 10 millimeters in diameter. In cross section, upward turning of the muscular coat was seen. At the cardiac and the posterior wall edge there was a soft, white nodule, the size of a pea, just beneath the mucosa. Six blocks were removed for histological examination.

Histology. The ulcer base consisted of a dense, cicatricial connective tissue, covered with a superficial necrotic layer. At the anterior edge of the ulcer wall the muscularis mucosæ and the muscular coat were fused. Figures 13 and 14 present a most typical picture. Near the edge of the ulcer the glands become highly irregular and push deeply stained processes into the submucosa. The ulcer

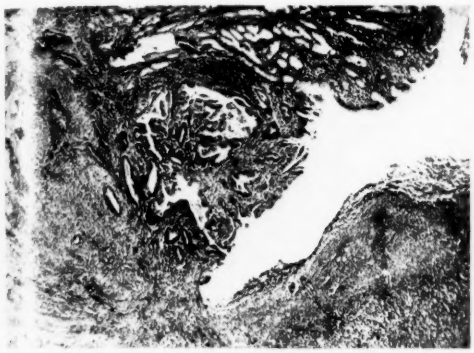

Fig. 15. Case 6. Small adenocarcinoma embedded within the mucosa at the edge of the ulcer. ×27.

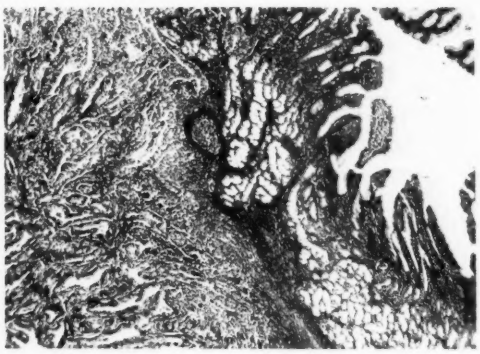

Fig. 16. Case 6. Irregular glands invading the submucosa. ×27.

base itself contained no atypical glands. All other blocks showed a broad zone of typical adenocarcinoma with medullary parts growing not farther than 5 millimeters into the ulcer base.

CASE 6. A woman, aged 46 years, complained of severe epigastric and right upper quadrant pains radiating into the shoulders, and frequent bilious vomiting, of 1 year's duration. She had had a fair appetite and had not lost weight during her illness. At clinical examination only deep epigastric tenderness was found; no abnormal mass could be palpated in the abdomen. Test meal yielded free hydrochloric acid, 10; total acidity, 28. X-ray examination of the digestive tract failed to disclose any abnormality. No visualization of the gall bladder could be obtained through intravenous dye administration. In spite of internal medication her complaints grew worse, so laparotomy was performed. A rigid, scar-like mass was found on the lesser curvature near the pylorus. The distal half of the stomach was resected. In the operative specimen a prepyloric round ulcer, about 30 millimeters in diameter, was found. It had soft, raised, somewhat overhanging edges, especially at the cardiac edge. Its floor consisted of a smooth, firm, whitish tissue. In cross section the muscular coat was found to be turned upward. The mucosa showed marked redundance and was whitish in color all around the ulcer margin, except the pyloric edge, where it looked like a common callous ulcer. Four blocks were removed for microscopical study.

Histology. The ulcer floor was formed by the granulation tissue becoming highly fibrous toward the depth and was covered by a superficial necrotic layer. In the connective tissue, heavily infiltrated—chiefly by eosinophils—there were many large lymphatic nodules with well developed germinative centers. At the ulcer edge the muscular coat radiated into the ulcer base, where it was lost. The muscularis mucosæ and the muscular coat were separated by a rather broad band of cicatrized submucosa, except at duodenal edge, where they were approximated, almost fused. No endarteritic vessels in base.

In the block taken from the pyloric edge, just at the ulcer margin, a round area about 2 millimeters in diameter was embedded within the mucosa. It consisted of irregularly arranged glandular structures (Fig. 15). The glands were lined with 2 to 3 layers of epithelial cells showing marked polymorphism. The majority of the cells were dark with deeply stained nuclei; there were, however, parts where the cells were rather pale, vacuolar. Very few mitotic figures were seen. No transition between this area and the normal mucosa was observed. About 5 millimeters from this area another quite similar nodule lay also entirely within the mucosa. In all other blocks the ulcer margin was formed by a broad zone very similar to the atypical glandular tissue described. At the cardiac edge many irregular glands invaded the submucosa (Fig. 16). In the other blocks the neoplasm did not penetrate the muscularis mucosæ nor were glands anywhere encountered in the ulcer base. In some parts the tumor produced many papillary projections lined with 2 to 4 layers of epithelium showing moderate polymorphism. Other projections consisted merely of epithelium, with connective tissue axis. The latter had a highly polymorphous epithelial covering with many mitotic figures. Parts of the neoplasm were necrotic and infiltrated with polynuclear leucocytes.

We consider this case a carcinoma arising from an adenomatous hyperplasia in the edge of a chronic gastric ulcer.

SUMMARY

We do not wish to draw conclusions from our statistics as our material is relatively small, but we would like to call attention to the fact that cancer secondary to ulcer is not the rarity it is believed to be and taught by many authors. In 64 cases of chronic peptic

ulcer and 26 cases of carcinoma, we had 6 cancers secondary to ulcer. This fact justifies the opinion now held by the vast majority of prominent surgeons that whenever possible chronic gastric ulcers must be resected.

1. There are many discrepancies in the statistics as to the frequency of cancer developing on a peptic ulcer base.

2. Such cancers can be definitely diagnosed only by microscopic examination. Diagnosis is based on signs characteristic of chronic peptic ulcer and in addition there is found partial or complete freedom from cancer cells at the ulcer base and ulcer margin.

3. At the Third Surgical Clinic of the University of Budapest, in 64 cases of chronic gastric ulcer and 26 cases of carcinoma, there were 6 cases in which cancer developed on an ulcer base.

4. Because carcinoma relatively frequently arises in peptic ulcer, such ulcers should be resected whenever possible.

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CARCINOMA OF THE MALE BREAST

With Special Reference to Etiology[1]

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THE purpose of this paper is to present a clinical and pathological study of 47 cases of male breast cancer with a summary of two postmortem reports. Forty-one cases are reported for the first time while 6 of the Memorial Hospital cases have been reported in part by Wainwright (Cases 1, 2, 3, 19, and 20) and Coley (Case 21). An attempt is made to correlate the etiological factors especially gynecomastia in male breast cancer with those of cancer of the breast in women.

The early history of the recognition of this disease is of interest. In the writings of Franciscus Arcaeus (1493–1573) we find the earliest reference, in that it is stated that cancer occurs also in males although not so frequently as in females. Fabricus Hildanus (1537–1619) described a single case of male breast cancer while Louis Heister, the famous German surgeon, described male mammary cancer at length in his inaugural dissertation. After reference to the publications of his time Morgagni (1682–1772) described several observations of his own regarding this disease. The frequently cited case of Thomas Bartholinus (1616–1682) probably refers to mammary cancer in a woman (Wolff).

About a century later the theses of Horteloup (1872) and of Poirier (1883) were the first to record systematic studies of this disease. Schuchardt followed Poirier a year later with two elaborate reports mostly from German and Austrian sources. The subject was first summarized in the English literature by Williams (1894) and Warfield, who reviewed the known cases and added observations of their own. Recently Wainwright (1927) made an extensive review of the literature and added new material; he reported on 418 cases with an examination of the pathological material in 79.

Incidence. At the Memorial Hospital, Pack and Le Fevre found male breast cancer comprised 1.24 per cent or 0.41 per cent of all cancers in males. Deaver and McFarland found that 1.5 per cent of all mammary cancers occurred in men, while Schuchardt stated that of all malignant breast tumors about 2 per cent occurred in the male. According to several authors the percentage varies from 0.86 to 8.4; probably this latter figure is too high if a large series of cases of both sexes is carefully studied. The Census Bureau of England and Wales in 1926 reported on 5,339 mammary cancers of which only 0.08 per cent were in the male. Cancer of the male breast occurred only 9 times in 950 cases between the years 1889 to 1931 in the Johns Hopkins Hospital series.

ETIOLOGY

Heredity. It appears that heredity is of secondary importance as a causative factor in male breast cancer. Several of the older patients who were foreign born made it impossible to ascertain an accurate history in regard to heredity and in the remaining cases no definite family history was obtainable. Four of Judd's 17 cases and 2 of 11 cases of Finsterer's series gave a positive history of cancer. Von Winniwarter considered heredity a causative factor in 5.8 per cent.

Sex factor. Many explanations are given to account for the relative rarity of cancer of the breast in men as compared with women. The development of the gland is similar in both sexes until puberty. In the female, the course is then one of great functional activity and cyclic changes, whereas in the male the gland remains relatively stationary with less variation in the anatomical structure. The response to endocrine stimuli will be discussed later in the paper. However, this inherent difference in the function of the gland is the most commonly accepted explanation. The rare occurrence of cancer of the male breast is in accord with the general rule that cancer seldom arises in vestigial structures.

Age incidence. It is a general opinion of observers since Poirier, that cancer of the breast develops at a later age period in males than in females. It has been said that women

[1] From the Breast Clinic of the Memorial Hospital, New York.

Fig. 1. Case 39. H. A., aged 50 years. Marked bilateral gynecomastia with cancer of the left breast. Clinical group, primary operable.

Fig. 2. Case 20. M. R., aged 37 years. Bilateral hypertrophy with cancer of the left breast. Clinical group, primary inoperable.

age earlier than men and it is a striking fact that 33 of 47 patients, or 70 per cent of this series, were over 50 years of age. Wainwright found the average age to be 54.2 years in 401 cases. The oldest reported case was 91 years of age (Lunn), whereas the youngest patient was 12 years of age (Blodgett). The ages at the time of observations in cases in the present report are shown in Table I.

TABLE I.—AGE INCIDENCE

	Number of cases	Per cent
30 to 39 years	7	14
40 to 49 years	7	14
50 to 59 years	17	35
60 to 69 years	12	25
70 to 79 years	3	6
80 to 89 years	1	2

Of 47 patients the oldest was an 83 year old negro, while the youngest was 31 years of age. The average age of this group was 54.4 years, whereas the mean age was 57 years.

Color. Two of the American born patients were negroes. Wainwright mentions that numerous cases of mammary cancer in negroes are reported by American surgeons but records no relative percentages. Lewis and Rienhoff report 3 negro patients in their series of 9 cases.

Side involved. The present series shows left breast involvement in 26 patients, the right breast in 20, and bilateral involvement in one patient. The predominance of the left side appears to coincide with the findings of various authors. Lane-Claypon in a recent statistical summary reported a slight left sided preponderance in women.

One patient, L. G., Case 32, had bilateral involvement. The primary tumor was

Fig. 3. Case 35. A. M., aged 54 years. Moderate hypertrophy of the right breast. The left breast is replaced by cancer. Clinical group, primary operable.

Fig. 4. Case 34. O. McN. Primary operable cancer of the right breast. Note the small disc-like mass with early ulceration.

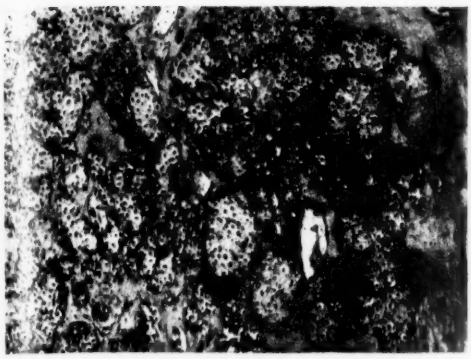

Fig. 5. Photomicrograph showing the cell structure in Case 34. (See Fig. 4.) Alveolar carcinoma simplex, grade II, radioresistant.

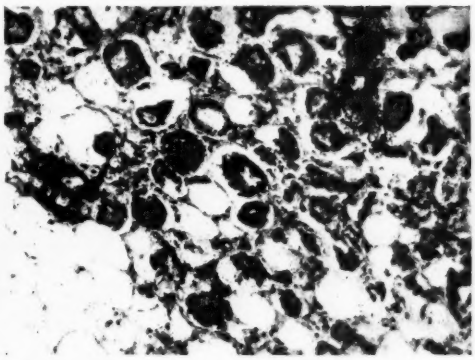

Fig. 6. Case 36. R. P., aged 56 years. Adenocarcinoma, grade II, radioresistant. This patient presented moderately hypertrophied breasts.

noted in the left breast and removed surgically at another hospital. He was then referred to the Memorial Hospital for postoperative irradiation. One year later the patient developed a firm, nodular mass beneath the nipple of the right breast, 2.5 by 2.5 by 1 centimeters in diameter associated with involvement of the axillary lymph glands.

Some observers have suggested that inasmuch as the majority of people are right handed, the right side of the body might receive more trauma than the left, a theory not borne out by the facts if trauma is a cause. Wainwright in a total of 336 cases showed the right side involved 163 times, the left 170 times, and, in 3 patients, both breasts.

Previously existing benign tumor. Three cases, or 6.3 per cent, in the present series gave a history of having a tumor removed from the breast prior to the onset of malignant growth. Only in the following case was the benign tumor re-examined in this laboratory. Patient 20 had a growth removed from the left breast diagnosed as sweat gland adenoma 15 years prior to the development of a painful mass in the same breast. On examination a small fixed tumor mass of the breast and several enlarged nodes were present in the axilla, as well as multiple skin nodules. A biopsy of the second tumor showed fibrocarcinoma simplex grade II.

We wish to stress the fact that any small localized swelling of the male breast with or without classical signs of cancer should be considered as malignant until proved otherwise by microscopic examination. Patients who present themselves with disc or button-like indurations beneath the nipple should have surgical removal performed at once.

Wainwright, in reporting the details of 5 cases of previously existing benign tumor with an alleged duration of from 20 to 24 years, was able to study only 3 of the microscopic sections; but in none of these was histological confirmation of the existence of previous benign tumor obtained.

Schreiner believes that chronic inflammatory lesions often precede the development of a malignant tumor, and it is for this reason

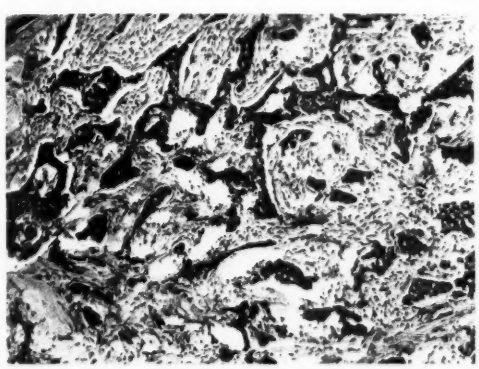

Fig. 7. Case 23. E. K., aged 79 years. Fibrocarcinoma simplex, grade II, radioresistant.

that benign tumors in the male breast are treated by radical amputation. Other observers are less radical and believe that local mastectomy followed by irradiation treatment will control cancer of the male breast.

Trauma. Previous history of injury was obtained in 14 or 29 per cent of the cases in this series. A single severe injury was reported in 12 of the 14 cases, while 2 patients were accustomed to exert pressure against the chest wall while following their usual trades. It should be noted that in all these cases the previous integrity of the breast and the authenticity and sufficiency of the trauma rest entirely upon the statements of the patients and were not verified. That the tumors arose at the exact point of the injury was also not determined.

It is important to obtain a critical history of the sequence of events in those instances in which a single severe trauma is apparently followed by the development of cancer. Ewing clearly states the factors which should be observed in making a positive judgment of the relationship of trauma to breast tumors. He insists that such relationship may be assumed to have causal significance, only if the breast can be shown to have been previously normal and the injury to have been severe enough to have caused interstitial hæmorrhage and solution of continuity of the breast ducts; also that there must be some indication of continuity of symptoms between the trauma and the appearance of the tumor and that even in those cases one can only maintain a probable relationship.

Knox also warns us that it would be as inaccurate and unscientific to ascribe the origin of a cancer to a single blow, as it would be to judge the duration of a tumor of the breast from the patient's statements.

Murphy on the other hand makes a radical statement that the breast is the only organ in the body where cancer will develop following a single mild trauma. Concerning the influence of trauma, reports in the literature vary greatly. Schuchardt considered 25 of his 219 cases due to contusion or mechanical causes. In only one of Judd's series of 17 cases was trauma mentioned as a possible causative factor. Wainwright reports the relationship so frequently that he feels it must be taken into consideration as a cause of cancer at least in the male breast.

Gynecomastia. While the observation of cancer with gynecomastia has been noted in occasional case reports, it has not been sufficiently emphasized. Ewing states that unusual development and activity of the breast are predisposing conditions for cancer of the male breast.

Pertinent facts in regard to the complex hormonal control of breast development by means of pituitary, thyroid, prostatic, and testicular secretions may be summarized as follows:

Experimental evidence. Two types of experimental procedure—organ transplantation and the use of organ extracts—have been used to show the relation of the ovary to new development of the mammary gland. Athias and later Steinach were able to produce breast growth even in male guinea pigs by the implantation of ovaries. Ferguson and others have demonstrated that following daily injections of prolan A in laboratory animals over long periods of time an epithelial hyperplasia of the prostate is noted. An imbalance between the testis and pituitary must account for the production of a small relative increase in the pituitary secretion which acts directly upon the prostate and indirectly upon mammary tissue. Furthermore, if the testes are removed, atrophy of the prostate follows; showing that the pituitary substance must act through the testes. Loeb points out that with regard to the arrested growth of the mammary gland it is possible that the developing testis may exert a certain inhibiting effect.

Clinical evidence. Taylor showed by clinical observations that the breast epithelium of the female is normally responsive throughout life to stimulation by the internal secretion of the ovary. The literature is replete with instances of gynecomastia associated with imperfect development of the male sexual organs, such as pseudohermaphroditism, after atrophy and removal of the testes, and associated with malignant testicular tumors. We have observed the association of excessive secretion of prolan A in the urine of patients with teratoma testis associated with gynecomastia.

Typical gynecomastic breasts were noted in 5 such patients and even breast secretion in one instance. At autopsy an excessive proliferation of the epithelium of the prostate was also regularly observed.

In patient 23 there was disclosed at autopsy a persistent thyroglossal duct and bilaterally undescended fetal testes. These fetal abnormalities involving the thyroid and the testes were possible sources of endocrine imbalance in this patient. A young man was recently observed at this hospital presenting gynecomastic breasts as well as typical evidence of exophthalmic goiter. Many patients with gynecomastia reported in the literature have had other endocrine disorders which might influence the testis and thus be indirectly related to hypertrophy of the male breast.

Co-existent gynecomastia with cancer. Mammary hypertrophy has been studied by Pigot, Schuchardt, Berns, Imbert, and Villeon who report cases of concurrent cancer and gynecomastia. Imbert and Collignon strongly considered gynecomastia as an etiological factor in certain cases of cancer of the breast.

Berns reports an unusual case from the standpoint of the size of the hypertrophied breast. A 42 year old man stated that since youth both breasts had been enlarged. He recalled a blow to the right breast received in childhood and followed by acute pain for several days. No secretion from the breast was ever noted. He sought consultation in September, 1884, because of a small tumor of long duration in the right breast. Both breasts were extraordinarily developed, resembling the female type, pendulous, and containing fatty tissue. The horizontal diameter of the left breast was 17 centimeters and of the right 19.5 centimeters. The vertical diameter of the left breast was 14 centimeters, and of the right 16 centimeters. The right breast was the seat of an oval tumor, 6 by 9 centimeters in diameter located about 2 centimeters from the nipple. The mass was freely movable over the pectoral muscles and no involvement of the axillary gland was noted. The right breast as well as the normal left breast were surgically removed in November, 1884. A second operation was done for local recurrence in May, 1885, and another local recurrence was removed a year later. The patient died in May, 1886, with signs of pulmonary œdema (metastasis?). Microscopic examination of the breast tumor revealed an alveolar scirrhous cancer of the breast.

Villeon's case demonstrated the not uncommon occurrence of gynecomastia associated with benign tumor of the prostate, with a later development of cancer in one breast. A 75 year old man was operated on 12 years previously for benign hypertrophy of the prostate. Following this operation he developed bilateral gynecomastic breasts, which were the type and shape of the female gland. Later a typical cancer of the right breast developed with nipple retraction and axillary gland involvement. Five years after mastectomy local recurrence was noted which was treated by roentgen therapy. The microscopical diagnosis of the breast tumor was epithelioma.

Anatomical evidence. That the male breast contains abundant glandular tissue and not merely adipose tissue, as was previously supposed, has been demonstrated by Bailey, Andrews, and Kampmeier and more recently by von Gusnar. He examined 106 male breasts at various ages and found in gynecomastia that the connective tissue is only slightly increased while the glandular tissue shows a marked hyperplasia; and concluded that such breasts are more liable to be the seat of cancer. Wainwright (p. 843) shows a large cross section of such a gland and states that it is a frequent type in his experience. Hinze removed a hypertrophied breast weighing 125 grams which showed areas of glandular tissue on microscopical examination.

Summary of present series of gynecomastic cases. We have observed 9 patients, or 19 per cent of the entire series, who presented hypertrophied mammary glands, corresponding in general topography to the female breast. All of this group were white and ranged from 37 to 73 years. The left breast was involved with cancer 6 times and the right breast, 3 times. Trauma was mentioned in the antecedent history only 4 times.

The shortest duration of the existence of the tumor before examination at the clinic

was 3 months, while the longest interval was 48 months. Pain was noted twice, bleeding once, nipple retraction 9 times, and ulceration 9 times. For clinical grouping the patients were classified as primary operable, 3; primary inoperable, 5; and recurrent inoperable, 1. The disease was markedly advanced in the majority, 66 per cent being inoperable on admission.

The types of cancer observed were adenocarcinoma, 4; duct carcinoma, 2; and carcinoma (punch aspiration), 2. The re-examination of tissue was not possible in one instance. Of 5 sections available for grading, 4 were grade II and 1 was grade I. Corresponding to the clinical grouping, metastases were distributed as follows: axilla, 8; supraclavicular, 6; lungs, 4; and bones, 1.

Surgical procedures were carried out in 5 patients. Local and radical mastectomy were each performed twice and in one other patient gold radon tubes were implanted in the tumor.

Five patients are dead while 2 are alive with evidence of cancer. Two are alive without evidence of disease 11 months and 18 months, respectively, from the time of first observation.

SYMPTOMATOLOGY

Such symptoms as pain, bloody discharge from, and retraction of, the nipple varied according to the extent of the disease and the histological structure of the tumor.

In all cases the patient noted the presence of a tumor mass. In 14 patients the tumor mass was the first sign to call attention to the disease. The majority of the tumors were in the region of the nipple, which agrees with observations made by Speed, who reported that in 50 per cent of his cases the nipple becomes involved. Because of incomplete histories and the number of recurrent cases treated in this series, it is impossible to determine the quadrant originally involved.

Other symptoms were noted as follows: pain, 13 times or 27 per cent; bleeding, 4 times or 8 per cent; nipple retraction, 14 times or 29 per cent; and ulceration, 14 times or 29 per cent. Figure 4 shows the type of ulceration found with a relatively small tumor mass. Extensive ulceration is common since the growth is near the skin and extends to involve it by direct continuity. Fourteen patients gave a history of previous scab formation or ulcerations. Ulceration seemed to bear no relation to the duration or the size of the tumor; it is common and tends to appear early. Ulceration occurred in all types of tumors of the male breast observed in this study and yet no instances of typical Paget's disease or sweat gland cancer, which in the female breast are commonly associated with early ulceration, were seen.

Clinical classification. Of the total series the patients were classified on admission as follows: Primary operable 18, primary inoperable 10, and recurrent inoperable 19. Twenty-nine patients, or 61 per cent, were inoperable.

PATHOLOGY

In any consideration of the normal histology of the male breast, the fact that glandular tissue is consistently noted is of importance in the physiology and pathology of this organ. Bailey, at the London Hospital, upon examination of male breasts of postmortem subjects between the ages of 6 months and 65 years, found glandular tissue present in all cases. Andrews and Kampmeier found a complex series of ducts and alveoli in every male breast examined. Their description is quoted in detail, since the histology of this organ is given but scanty attention in most textbooks of anatomy. "The ducts are lined by a single layer of columnar epithelium and are invariably patent. They lie imbedded in rather dense connective tissue. The capsule is not sharply defined and blends with the adjacent fibrous tissue. There is a rather sharply differentiated periductal tissue which consists of less deeply staining fibers containing a greater number of nuclei. This periductal zone bears a close resemblance to growing tissue and has been mistaken for proliferating fibrous tissue. Investigations show that probably in the majority of adult males, the breast persists in essentially the same state as in the preadolescent female, and there exists in the male breast a complicated system of open ducts which are far from being vestigial in nature."

Forty-one tumors available for re-examination were classified by Ewing as in Table II.

TABLE II.—EWING'S CLASSIFICATION OF 41 TUMORS

Type of tumor	Cases
Carcinoma simplex	25
Adenocarcinoma	9
Scirrhous (fibro) carcinoma	2
Epithelioma	1
Carcinoma (punch aspiration)	5
No tissue	5

Thirty-two sections were graded as follows:

Grade (differentiation)	Cases
Grade I	1
Grade II	24
Grade III	7
Not graded	15

Twenty-five of 42 sections, or 59 per cent, available for review, were classified as carcinoma simplex. Wainwright found that carcinoma simplex is less common in men, reporting only 3.7 per cent in his series of 78 cases; but Warfield, on the other hand, agreed with the findings in the present series that carcinoma simplex was the histological diagnosis most frequently made.

With one exception, all tumors classified as carcinoma simplex were grade II and radioresistant. The remaining case was a very cellular grade III radiosensitive tumor. There were no grade I tumors in this group. Of the whole series only one tumor was of grade I degree of differentiation, while 7, or 21 per cent, were grade III. While a determination of the degree of radiosensitivity is a difficult problem, in 18 of 20 sections diagnosed as probably radioresistant, the clinical course after thorough irradiation proved fatal. In one patient, Case 29, a cellular carcinoma simplex, grade III, radiosensitive, entirely disappeared following one cycle of X-ray therapy.

A striking comparison between Greenough's group of women compared with an almost equal group of men studied by Wainwright shows that in both groups the number of cases in each assigned to the low and high malignancy classes is practically equal.

A summary of the 4 cases in the high group (grade III) is of interest.

CASE 37. B. H. had noted a tumor mass 18 months previous to admission. He died 4 months following first observation making a total duration of the disease 22 months following the first symptoms.

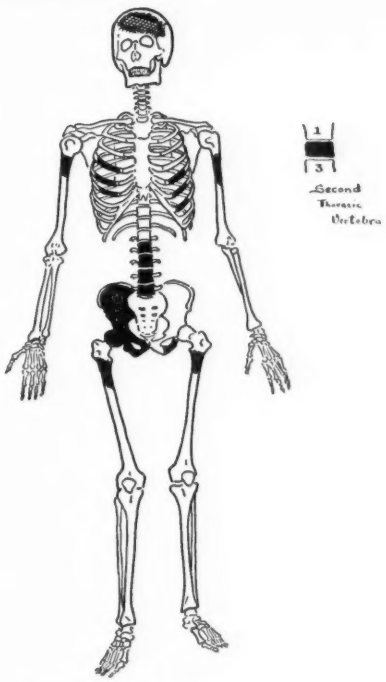

Fig. 8. Case 30. S. R., aged 31 years. Negro. Recurrent inoperable cancer of the left breast. Diffuse bone involvement indicated by shaded areas. Pathological fractures of the left humerus and of the right femur. Total duration of disease 5 years and 2 months.

CASE 44. M. S. had noted a mass in the left breast 18 months previous to admission. He lived 2 months following first observation in the clinic making a total duration of the disease of 20 months.

CASE 41. O. K. had noted a mass in the left breast 8 months previous to admission. He lived 15 months following first observation making a total duration of the disease 23 months.

CASE 30. S. R. had noted a mass in the left breast late in 1919. A radical mastectomy was performed in August, 1923, at another hospital. Because of pain in the right lumbar region he was referred to the Memorial Hospital for diagnosis and treatment. His condition at time of death was one of extensive involvement of practically all the long bones of the body with pathological fractures of the left humerus and the right femur. The right innominate bone was practically replaced by cancer (Fig. 8). He was transferred to the United States Marine Hospital where he died May, 1925, making a total duration of life from the onset 6 years and 3 months.

TABLE III.—SUMMARY OF EIGHT CASES TREATED BY IRRADIATION ONLY

Case No.	Age	Clinical group	End-result	Length of life following first observation
L. W.[9]	50	Primary operable	Alive 7-20-1932 without disease	11 mos.
C. K.[15]	61	Primary inoperable	Alive 11-21-1932 with disease	9 mos.
W. C.[18]	68	Primary inoperable	Died 3-22-1932 with axillary supraclavicular and lung metastases	7 mos.
J. D.[29]	83	Primary operable	Died 7-14-1928 of cerebral hæmorrhage	6 mos.
A. M.[35]	54	Primary inoperable	Died 6-5-1927—Skin and bilateral axillary metastases	8 mos.
H. A.[39½]	50	Primary inoperable	Died 1-20-1920—Axillary and supraclavicular metastases	7 mos.
F. S.[45]	54	Primary inoperable	Died 4-15-1923—Bilateral axillary and pulmonary metastases	5 mos.
M. S.[46]	55	Primary inoperable	Died 2-15-1926—Metastases to axilla, supraclavicular fossa, lungs and bones	14 mos.

Summary of two cases with autopsy findings:

CASE 22. J. A. K. age 65, white, married, Jewish. Family history negative, past history negative. The patient was struck by the corner of a mail car rack in 1915 at the upper outer quadrant of the right breast. In 1916, he noted a small mass at the site of the alleged injury. Local medication failed and mass increased to the size of a quarter. The tumor showed moderate fixation to the chest wall with evidence of right axillary and right supraclavicular metastases. The left breast and axilla were normal.

Radical mastectomy was performed July 13, 1917. The microscopic diagnosis was adenocarcinoma, grade II, radioresistant. A chest plate May 14, 1923, showed evidence of bilateral pulmonary metastases. One month later he fell unconscious and thereafter vomited practically everything taken by mouth. A fluoroscopic examination of the stomach at another hospital was reported negative. He was admitted to Memorial Hospital July 15, 1924, in extremely poor condition, rapidly lost weight and strength and died August 18, 1924, 8 years from the onset of the first symptom.

Microscopic diagnosis of autopsy material: lungs, cellular alveolar carcinoma; axillary nodes, free; stomach and liver, carcinoma resembling the primary tumor of the breast.

CASE 23. E. K. age 79, single, white, Bohemian. Family and past history are not contributory. About 3 years ago the patient was struck in the region of the left nipple by a piece of wood splinter, but no bleeding followed this injury. Three months after the trauma he noted a small ulcer in the region of the left nipple which gradually spread over the entire breast area and became infected. He did not consult a physician for 2 years, during which time he developed marked swelling of the arm and numerous skin nodules over the entire thorax. For the last 3 months he had lost strength and developed some cough but no hæmoptosis.

On examination the patient appeared very weak with evident loss of weight. An ulcerated lesion over the region of the left breast consisted of an irregular area about 6 centimeters in diameter, with raised and indurated edges. There was marked non-specific elephantiasis of the left arm. Several hard nodes in the supraclavicular area were noted and the body was covered with numerous subcutaneous nodules varying from 1 to 2 centimeters in diameter. A biopsy December 16, 1927, showed carcinoma simplex, grade II, radioresistant. A diagnosis was made of primary inoperable cancer of the left breast with axillary, supraclavicular, and skin metastases, and probably also pulmonary involvement.

He was admitted to the Montefiore Hospital January 11, 1928, *in extremis*, and died suddenly the following day. At autopsy, the left breast was found to be the seat of a tumor mass with numerous metastases to the skin, lungs, bronchial, axillary, inguinal, and retroperitoneal lymph nodes, thrombosis of the axillary veins with marked non-specific elephantiasis of the left upper extremity; chronic passive congestion of the liver; persistent thyroglossal stalk and bilaterally undescended fetal testes; generalized atherosclerosis and arteriosclerosis of the kidneys; organized thrombosis of deep femoral arteries, bronchopneumonia of the left lower lobe, and acute splenic enlargement. The development of non-specific elephantiasis of the left arm without previous operative procedure is to be noted. The autopsy findings of a persistent thyroglossal stalk and bilateral undescended testes is significant as a possible etiological factor.

Multiple tumors. A single instance of multiple tumor occurred in Case 33, a 43 year old man who presented a recurrent inoperable

cancer of the right breast, as well as multiple lipomata of the extensor surface of the right forearm, the left upper arm, and of the skin of the chest wall.

METASTASES

All patients known to be dead or lost to record showed clinical or X-ray evidence of metastases usually multiple and widespread. Of 16 patients alive at time of present report, 9 have no evidence of recurrence or metastatic disease; however, only 3 of the 9 patients were observed before January, 1928. Axillary lymph nodes were involved in 10 of 23 patients at the time of radical operation. The distribution of metastases was as follows: skin, 8; axilla, 30 (4 times bilateral); supraclavicular fossa, 17 (1 case bilateral); lungs, 21; bones, 5.

TREATMENT

Surgical. Thirty-eight, or 80 per cent of the series, had some surgical procedure while only 9 patients were treated by other methods. One patient had a local removal of the primary tumor in another hospital followed by radical mastectomy here. Radical mastectomy was performed 24 times, local mastectomy 8 times, and local removal of the tumor 6 times. As a majority of these patients received some form of irradiation therapy, the end-results will be discussed in one group.

Fourteen of Judd's 17 cases were treated by radical amputation. Wainwright showed that nearly half of the men apply for treatment during the first 12 months; in spite of these figures, which compare favorably with statistics of a similar kind in breast cancer in women, there is considerable delay between the onset of the disease and surgical or irradiation treatment. Various reasons are given to explain this apparent neglect. That the male often does not consider himself liable to cancer of the breast, and therefore has much less fear of the disease is perhaps the principal cause for the delay. In many instances when a small freely movable tumor is brought to the physician's attention, the condition is not given serious consideration until typical signs of cancer appear. Cancer of the male breast should be treated on the same principles as in the female. Biopsies should be taken for frozen section diagnosis, if there is any uncertainty concerning the true nature of the disease. That these tumors are often small does not justify limited removal and operative procedures should be radical regardless of the desire to avoid impairment of arm function.

Five patients, or 10 per cent of this series, developed non-specific elephantiasis of the arm. Treves discussed this complication as seen with cancer of the breast in women, and his conclusions in regard to the management of these patients applies equally to the male breast cases. None of our patients received surgical treatment for this condition.

Radiation. All 16 patients under present observation have received some form of irradiation treatment, while 23 of 28 patients now dead were so treated. These patients were treated by a variety of methods, and for that reason an evaluation of end-results in this group is without definite value. The technique varied from a single exposure of low voltage, unfiltered X-rays to repeated courses of treatment with both the radium element pack and high voltage X-rays up to full erythema dosage.

The cases under present report have been treated over a period of many years. In the earlier years we had nothing but low voltage X-ray, and the dosage could not be checked with accurate measurements now available. Such treatments delivered to the tumor only a fraction of an erythema which we know now was inadequate to destroy breast cancer. With our present knowledge such patients, classified according to their clinical status and

TABLE IV.

Case No.	Age on admission	Date of mastectomy	Type and grade of tumor	Last note	Survival period
[1] G. L.	57	2–3–1923 Radical	Carcinoma simplex, grade II	5–21–1930 free of disease	7 yrs. 3 mos.
[2] F. A.	74	9–5–1925 Local	Fibro-carcinoma simplex, grade II	8–29–1932 free of disease	6 yrs. 11 mos.
[3] S. A.	64	9–22–1921 Radical	Cellular adeno-carcinoma, grade II	11–12–1930 free of disease	9 yrs. 2 mos.

TABLE V.—ANALYSIS OF FORTY-SEVEN CASES OF CANCER OF

Case No.	Date of admission	Age	Color	Side involved	Clinical group	Trauma alleged	Gynecomastia	Type of tumor	At operation—Axillary nodes involved	Grade of malignancy	Metastases				
											Skin	Axilla	Supraclavicular fossa	Lungs	Bones
1 G. E.	1- 8-23	57	White	Left	Primary operable	None	None	Carcinoma simplex	None	II	None	None	None	None	None
2 F. A.	9- 4-25	74	White	Right	Primary operable	None	None	Fibrous carcinoma simplex	None	II	None	None	None	None	None
3 S. A.	9-12-21	64	White	Right	Primary operable	Yes	None	Cellular adenocarcinoma	None	II	None	None	None	None	None
4 A. B.	4- 3-29	52	White	Left	Recurrent inoperable	None	None	Carcinoma simplex	None	II	Yes	None	None	None	None
5 W. C.	7- 7-30	52	White	Right	Primary operable	Yes	None	Tubulo-alveolar carcinoma	None	II	None	None	None	None	None
6 S. B.	7-10-30	45	White	Left	Primary operable	None	Yes ✓	Small cell duct carcinoma	Yes	II	None	Yes	None	Yes	None
7 J. W.	10-20-30	65	White	Right	Primary operable	Yes	None	Carcinoma simplex	None	II	None	None	None	None	None
8 B. M.	5- 6-31	38	White	Left	Recurrent inoperable	None	None	Adenocarcinoma	None	II	Yes	None	None	Yes	None
9 L. W.	8-15-31	50	White	Left	Primary operable	Yes	Yes ✓	Carcinoma (aspiration)	None	Not graded	None	Yes	None	None	None
10 H. F.	6- 1-31	73	White	Right	Primary operable	Yes	Yes ✓	Duct carcinoma	None	II	None	None	None	None	None
11 S. A.	6-14-32	38	White	Right	Primary operable	None	None	Papillary adenocarcinoma	None	I	None	None	None	None	None
12 S. C.	11- 3-31	33	White	Left	Recurrent inoperable	None	None	Carcinoma simplex	Yes	II	Yes	Yes	None	Yes	Yes
13 T. G.	3-23-32	62	White	Right	Primary operable	None	None	Duct carcinoma	Yes	III	None	None	None	None	None
14 C. K.	5- 3-32	60	White	Left	Recurrent inoperable	None	None	Infiltrating carcinoma simplex	Yes	III	None	Yes	None	None	None
15 C K.	2-23-32	61	White	Right	Primary inoperable	None	Yes ✓	Adenocarcinoma	None	II	None	Yes	Yes	None	None
16 A. G.	9-19-30	38	White	Left	Primary operable	None	None	Basal cell epithelioma	None	Not graded	None	None	None	None	None
17 J. W.	6-24-31	45	White	Right	Recurrent inoperable	None	None	None	None	None	None	Yes	Yes	Yes	Yes
18 W. C.	8-12-31	68	White	Right	Primary inoperable	None	None	Carcinoma (aspiration)	None	Not graded	None	Yes	Yes	Yes	None
19 J.O'N.	9-16-20	52	White	Left	Recurrent inoperable	Yes	None	Adenocarcinoma	None	Not graded	Yes	Yes	None	Yes	None
20 M. R.	1-30-25	37	White	Left	Primary inoperable	None	Yes ✓	Adenocarcinoma	None	III	None	Yes	Yes	None	None
21 C. B.	11-20-27	67	White	Right	Recurrent inoperable	None	None	Adenocarcinoma	None	III	None	None	None	Yes	None
22 J. K.	9-20-19	65	White	Right	Recurrent inoperable	Yes	None	Adenocarcinoma	None	II	Yes	None	None	Yes	None
23 E. K.	9-16-27	79	White	Left	Primary inoperable	Yes	None	Carcinoma simplex	None	II	Yes	Yes	Yes	Yes	None
24 S. R.	2-18-29	55	White	Left	Primary operable	Yes	None	Infiltrating duct carcinoma	None	II	None	Yes	None	Yes	None
25 M. R.	5-16-24	53	White	Left	Primary operable	None	None	Fibrocarcinoma	None	II	None	Yes	None	Yes	Yes

THE MALE BREAST OBSERVED AT THE MEMORIAL HOSPITAL

Previous benign tumor	Previous duration in months	Symptomatology				Operation	Post-operative recurrence	Elephantiasis	Irradiation treatment	Last note	Five year survivals
		Pain	Bleeding	Nipple retraction	Ulceration						
None	9	Yes	None	None	None	2–3–23 radical	None	None	Yes	5–21–30 alive N.E.D.	Yes
None	12	None	None	None	Yes	9–5–25 local	None	None	Yes	8–29–32 alive N.E.D.	Yes
None	48	Yes	None	None	None	9–22–21 radical	None	None	Yes	11–12–30 alive N.E.D.	Yes
None	7	None	None	None	None	12–20–28 radical	Yes	None	Yes	7–24–31 alive N.E.D.	No
None	2	None	None	None	None	7–8–30 radical	None	None	Yes	3–29–32 alive N.E.D.	No
None	12	Yes	None	Yes	None	7–11–30 radical	None	None	Yes	11–11–32 Metastasis, lungs	No
None	84	None	None	None	Yes	10–27–30 radical	None	None	Yes	7–17–31 alive N.E.D.	No
None	18	None	None	None	Yes	1–30–30 radical	Yes	None	Yes	7–20–32 Metastasis, chest	No
None	4	None	None	Yes	None	None	None	None	Yes	7–20–32 alive N.E.D.	No
None	6	None	None	Yes	Yes	8–12–31 local	None	None	Yes	12–5–32 alive N.E.D.	No
None	5	None	Yes	None	None	7–6–32 local	None	None	Yes	9–26–32 alive N.E.D.	No
Yes	14	None	None	None	Yes	9–1930 radical	Yes	None	Yes	11–21–32 alive—pelvic recurrence	No
None	12	None	None	Yes	Yes	4–1–32 radical	None	None	Yes	11–14–32 alive N.E.D.	No
None	29	None	None	None	None	5–4–32 axillary dissection	Yes	None	Yes	11–11–32 alive N.E.D.	No
None	8	None	Yes	Yes	Yes	None	None	None	Yes	11–21–32 alive with recurrence	No
None	1	None	None	None	None	10–22–32 local	None	None	None	10–31–32 alive N.E.D.	No
None	8	None	None	None	None	1–15–31 radical	Yes	None	Yes	Died 4–22–32	No
None	48	None	None	Yes	Yes	None	None	Yes	Yes	Died 3–22–32	No
None	24	Yes	None	None	None	9–17–20 radical	Yes	None	Yes	Died 8–19–27	Yes
None	12	None	None	Yes	Yes	3–15–25 local	None	None	Yes	Probably died 3–8–27	No
Yes	72	None	None	None	None	4–25–25 radical	Yes	None	Yes	Died 2–15–29	No
None	36	None	None	None	None	7–13–17 radical	Yes	None	Yes	Died 8–16–24	No
None	36	None	Yes	Yes	Yes	None	None	Yes	None	Died 1–22–28	No
None	12	None	None	Yes	Yes	5–10–29 radical	Yes	None	Yes	Died 2–21–31	No
None	6	Yes	None	None	None	5–17–24 local	Yes	None	Yes	Died 12–19–31	Yes

TABLE V.—ANALYSIS OF FORTY-SEVEN CASES OF CANCER OF THE

Case No.	Date of admission	Age	Color	Side involved	Clinical group	Trauma alleged	Gynecomastia	Type of tumor	At operation—Axillary nodes involved	Grade of malignancy	Metastases				
											Skin	Axilla	Supraclavicular fossa	Lungs	Bones
26 J. S.	5-28-28	53	White	Left	Recurrent inoperable	None	None	Carcinoma simplex	None	II	None	None	Yes	Yes	None
27 A. C.	Not a hospital case 6-15-28	33	White	Left	Recurrent inoperable	None	None	Fibrocarcinoma simplex	None	II	Yes	Bilateral	Bilateral	None	None
28 C. M.	5-15-30	61	White	Left	Primary operable	Yes	None	Alveolar carcinoma	Yes	II	None	Yes	None	Yes	None
29 J. D.	2- 8-28	83	Black	Right	Primary operable	None	None	Carcinoma simplex	None	III	None	Yes	None	Yes	None
30 S. R.	3- 7-24	31	Black	Left	Recurrent inoperable	None	None	Carcinoma	Yes	Not graded	None	None	None	None	Yes
31 B. D.	9-25-26	46	White	Right	Recurrent inoperable	None	Yes ✓	Carcinoma	Yes	Not graded	None	Yes	Yes	Yes	None
32 L. G.	11-18-18	49	White	Bilateral	Recurrent inoperable	None	None	Scirrhous carcinoma	Yes	Not graded	None	Bilateral	Yes	None	None
33 A. K.	12-31-26	43	White	Right	Recurrent inoperable	Yes	None	Carcinoma simplex	Yes	III	None	Yes	None	None	None
34 O.McN	3- 5-27	52	White	Right	Primary operable	None	None	Alveolar carcinoma simplex	None	II	None	Yes	None	Yes	None
35 A. M.	10-22-26	54	White	Left	Primary inoperable	None	None	None	None	None	Yes	Bilateral	None	None	None
36 R. P.	12- 7-27	56	White	Left	Primary inoperable	Yes	Yes ✓	Fibro-adenocarcinoma	None	II	None	Yes	Yes	Yes	None
37 B. H.	7-15-18	65	White	Left	Recurrent inoperable	None	None	Carcinoma simplex	None	III	None	Yes	Yes	Yes	None
38 J. C.	1-20-19	61	White	Right	Recurrent inoperable	None	None	Carcinoma simplex	None	II	None	Yes	Yes	None	None
39 H. A.	6- 7-19	50	White	Left	Primary inoperable	Yes	Yes ✓	None	None	None	None	Yes	Yes	None	None
40 F. S.	12-31-19	59	White	Left	Primary operable	Yes	None	Carcinoma simplex	Yes	II	Yes	Yes	None	Yes	None
41 O. K.	3-19-20	44	White	Left	Primary inoperable	None	None	Alveolar carcinoma	Yes	II	None	Yes	None	Yes	None
42 T.McL	3-11-21	42	White	Left	Recurrent inoperable	None	None	None	None	Not graded	None	Yes	Yes	Yes	None
43 B. W.	11- 9-21	54	White	Right	Recurrent inoperable	None	None	Carcinoma simplex	None	II	None	Yes	None	None	Yes
44 M. S.	10- 6-22	50	White	Left	Recurrent inoperable	None	None	Alveolar carcinoma	Yes	II	None	None	Yes	Yes	None
45 F. S.	11-20-22	54	White	Right	Primary inoperable	None	None	None	None	None	None	Bilateral	None	Yes	None
46 M .S.	12-31-24	55	White	Left	Primary inoperable	None	Yes ✓	Adenocarcinoma	None	Not graded	None	Yes	Yes	Yes	Yes
47 E. K.	10- 6-31	66	White	Right	Primary operable	None	None	Carcinoma (aspiration)	None	Not graded	None	None	None	Yes	None

Abbreviation N.E.D. indicates "no evidence of disease."

MALE BREAST OBSERVED AT THE MEMORIAL HOSPITAL—Continued

Previous benign tumor	Previous duration in months	Symptomatology				Operation	Post-operative recurrence	Elephantiasis	Irradiation treatment	Last note	Five year survivals
		Pain	Bleeding	Nipple retraction	Ulceration						
Yes	84	None	None	None	None	1916, local removal	Yes	Yes	Yes	Died 7–20–30	No
Yes	12	None	None	None	None	1913 axillary dissection	Yes	None	None	Died 8–5–28	No
None	24	Yes	None	None	None	8–15–30 radical	None	None	Yes	Died 8–24–30	No
None	1	None	Yes	None	None	None	None	None	Yes	Died 7–14–28	No
None	48	None	None	None	None	8–28–23 radical	Yes	None	Yes	Died 5–23–25	No
None	48	None	None	Yes	None	8–15–26 radical	Yes	None	Yes	Died 1–14–30	No
None	10	None	None	None	None	5–5–18 radical	Yes	Yes	None	Probably died 4–20–20	No
None	20	Yes	None	None	None	1–14–26 local	Yes	None	Yes	Died 11–15–30	No
None	16	None	None	None	None	4–15–27 radical	None	None	Yes	Died 12–1927	No
None	120	Yes	None	None	Yes	None	None	None	Yes	Died 6–5–27	No
None	3	None	None	Yes	Yes	None	None	None	Yes	Died 6–22–28	No
None	6	Yes	None	None	Yes	10–1917 local	Yes	None	Yes	Died 12–1918	No
None	12	None	None	None	None	4–12–18 local	Yes	Yes	None	Died 2–1920	No
None	36	Yes	None	Yes	Yes	None	None	None	Yes	Died 1–29–20	No
None	72	None	None	None	None	1–15–20 radical	None	None	Yes	Died 8–25–21	No
None	8	Yes	None	None	None	3–16–20 bilateral axillary dissection	None	None	Yes	Died 7–1921	No
None	24	None	None	None	None	11–6–20 radical	Yes	None	Yes	Died Summer '21	No
None	72	None	None	None	None	1919 local	Yes	None	Yes	Died 12–1922	No
None	3	None	None	None	None	4–1920 radical	Yes	None	None	Died 12–1922	No
None	3	Yes	None	None	None	None	None	None	Yes	Died 4–15–23	No
None	4	None	None	Yes	None	None	None	None	Yes	Died 2–15–26	No
None	4	Yes	None	Yes	None	11–18–31 radical	None	None	Yes	Died 10–1–32	No

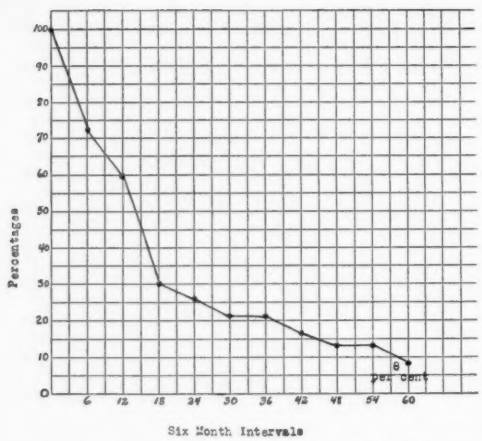

Chart 1. Percentage of survivals in *patients with disease* at six month intervals up to five years.

the histology of the tumor, should be treated with greater dosages.

Better knowledge, more varied and better modalities of treatment, together with the knowledge that the skin will stand heavier dosage than was previously supposed; and the anatomical position of male breast tumors, offers the radiologist a greater opportunity to cope with this disease. (Table III.)

As a means of relieving the constant pain of bone metastasis and as a method of treating local recurrences, irradiation should be recommended in these otherwise hopeless cases. In instances of radiosensitive tumors (Case 29), it can accomplish definite growth restraint in primary and metastatic areas.

Here also the advanced status of these cases is evident, as only 2 patients could be classified as early or primary operable. One patient observed too recently for end-result study has been treated by means of gold radon implants, while another patient, 83 years of age, was treated by X-radiation because of his age and general feeble condition. He died of cerebral hæmorrhage 6 months from the time of admission, free from local or metastatic evidence of cancer.

Judd believes that irradiation does not seem to have arrested the progress of the disease to any appreciable extent. Schreiner, in his series of cases treated by irradiation alone, had no early cases, and all but 1 are dead. This patient was treated for 2 years and 5 months and showed no evidence of recurrence. The 5 other patients are dead; 4 in less than a year, and 1 between 2 and 3 years.

END-RESULTS

Twenty-six patients observed prior to January, 1928, are considered in Table V. Three patients alive without evidence of disease 5 years or more following mastectomy and postoperative irradiation are tabulated in Table IV.

Two patients now dead lived over 5 years from the date of operation. Case 19, J. O'N., 52 years of age, on admission had noted a tumor mass of the left breast for 2 years. Radical mastectomy was performed September 11, 1920. He died of local recurrence and widespread metastases August 19, 1927, 6 years and 11 months after operation, and 9 years from the onset of the first symptom.

Case 15, J. K., 65 years of age, on admission had noted a tumor of the right breast 3 years prior to admission. Radical mastectomy was performed August 13, 1917, followed by postoperative irradiation. He died September 16, 1924, 7 years and 1 month following operation, from local recurrences and pulmonary metastases. He lived 8 years from the onset of the first symptom.

These cases emphasize the occasional long survival period in this disease and the necessity for frequent and careful re-examination. If such patients are lost to observation at the end of 5 years, they should not be considered as necessarily cured.

Eighteen patients, or 36 per cent of our total series, were classified as primary *operable* on admission. Ten patients, or 55 per cent, are now alive without disease, only 3 however were treated prior to January, 1928. One patient in the primary operable group lived 5 years, but subsequently died of recurrence. Another patient first observed in July, 1930, is alive with recurrence.

Ten patients, or 21 per cent, were classified as primary *inoperable*. No patients in this group lived 5 years either with or without disease. All patients are dead except one who now has recurrent disease.

Wainwright gives the details in his table (No. 6) of 20 patients dying more than 5 years after operation. In a later end-result study (1930) of 41 patients reported alive in April, 1927, Wainwright found 11 had since died—mostly of recurrences—while 4 patients could not be traced. Six of the 11 patients had passed the 5 year postoperative period. Twenty-six are alive at various periods, the longest interval being 18 years and 5 months. Of this group of 26 patients, 17 are alive more than 5 years after operation. Twenty-three, or 56 per cent of 41 cases, lived over 5 years after operation. However, it is not stated how many of these patients were free of disease at this time.

SUMMARY

1. Forty-seven cases of cancer of the male breast are reported, with an end-result study of 6 previously reported cases from the Memorial Hospital.

2. Male breast cancer comprised 1.24 per cent of admissions to the breast clinic and only 0.14 per cent of all cancers in males.

3. The average age of the patients in this group was 54.4 years.

4. The left breast showed a slightly higher percentage of involvement than the right.

5. "Occupational mastitis," due to chronic irritation, is not infrequently a precancerous lesion; however, a previously existing benign tumor was noted in only 3 patients, or 6.3 per cent of the series.

6. The incidence of trauma as qualified in the text is recorded as a possible etiological factor in 14 or 29 per cent of the cases. No *proved* instance of a single trauma causing cancer was noted in this series.

7. The coincidence of gynecomastia with cancer is emphasized, and evidence of their relationship is considered on experimental, clinical, and anatomical grounds. Gynecomastia occurred in 9 patients or 19 per cent of this series.

8. The symptomatology, pathology, and distribution of metastases corresponds to the well recognized manifestations of mammary cancer in general.

9. The prognosis of cancer of the male breast is poor. Five patients observed prior to January, 1928, survived 5 years. Three, or 11.5 per cent of these 26 patients, are still alive without evidence of disease.

10. Irradiation therapy is to be recommended as a valuable adjunct in the operable group and of great value as a palliative measure in the inoperable cases. Heavier dosage by irradiation methods is recommended.

I wish to thank Dr. Ewing for his review of the microscopical sections, and Dr. Lee for invaluable advice. I am indebted to Drs. Lee, Adair, and Treves of the Breast Clinic; and Drs. Coley, Craver, Quick, and Lenz for the use of their clinical records.

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PATHOLOGICAL AND CLINICAL DATA CONCERNING POLYCYSTIC KIDNEY[1]

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POLYCYSTIC disease of the kidney is discovered often enough on surgical exploration, or at necropsy, and occasionally on clinical examination, to warrant review of the pathological and clinical data which may be of aid in its recognition. If the condition is overlooked, it may be the cause of considerable confusion and may even lead to serious consequences. If, on the other hand, it is recognized, and thorough clinical examination is made, a fairly definite prognosis can be given, and the care of the patient may be materially altered.

The data in this article were collected from 193 patients observed at The Mayo Clinic, whose condition had been diagnosed polycystic kidney. Many of the patients returned for subsequent examination, and it was possible to corroborate clinical data previously obtained. Included in this group were 85 patients who were operated on for various renal complications occurring with polycystic disease, or whose renal condition was discovered in the course of operation for other abdominal lesions. Records of necropsy made at the clinic were available in 9 cases and operative specimens were obtained in 10 cases. Thus, histological studies were possible in 19 cases. Moreover, the findings at postmortem examination were available, in many of the cases in which death occurred after the patients had returned to their homes.

The incidence of congenital polycystic kidney found at postmortem examination at The Mayo Clinic was 9 in 9,171 cases, or a ratio of 1:1,019. The incidence noted clinically was 193 in 680,000 registrations, or 1:3,523.

PATHOLOGICAL DATA

The pathological anatomy of polycystic kidneys has been described repeatedly and only a few observations will be noted. As the cysts increase in size and multiply, the pelvis is encroached on and becomes deformed. The calyces may become elongated, and broadened, while others are abbreviated or even obliterated. This deformity can be visualized by means of pyelography. Polycystic kidneys are occasionally enormous; in our series, the largest were those of a man aged 40 years, who died of uræmia. Postmortem examination disclosed congenital polycystic kidneys each weighing 7,284 grams (approximately 17 pounds). The normal weight of a kidney is 300 grams. The left kidney measured 34 by 20 by 12 centimeters; the right, 33 by 19 by 12 centimeters.

Coincident cystic disease of the liver probably occurs more frequently than clinical data might suggest. In the 9 cases of polycystic renal disease in which postmortem examination was performed, associated cystic disease of the liver was found in 4, and of the pancreas in 1. Cystic disease of the liver is usually confined to limited portions of the organ, and is seldom the cause of symptoms suggestive of hepatic disease. Cysts of the liver do not usually attain large size, although in 1 case observed the cysts protruded to such an extent from both sides of the liver that they were confused with polycystic renal enlargement. Hepatic cysts are occasionally observed without any accompanying renal involvement. Other organs which have been reported to be involved in cystic degeneration are the ovary, broad ligament, uterus, bladder, and epididymis. This associated cystic degeneration would indicate a single origin of the condition, and strengthens the belief that it has a congenital basis.

Polycystic kidney is always bilateral in the adult. Even though, on intra-abdominal palpation during the course of operation, one kidney does not seem to be involved, it will

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subsequently prove to be cystic. In our series, in only 3 cases did renal enlargement appear to be unilateral at surgical exploration, and in 1 of these, enlargement of the other kidney developed subsequently. Clinical and surgical reports of unilateral polycystic kidney are of little value unless confirmed by careful postmortem examination.

The relation of polycystic kidney to the condition of the solitary cyst of the kidney is not entirely clear. It would seem probable that the solitary cyst is an acquired rather than a congenital lesion. Occasionally multiple scattered solitary cysts are observed, but seldom are they so numerous or of such character as to be confused with polycystic kidney. Solitary cysts are usually unilateral, although bilateral occurrence has been reported. Other types of cysts are those frequently found in arteriosclerotic kidneys, and the small, simple cysts that are found in kidneys that otherwise are apparently normal. These cysts are probably the result of localized inflammation or obstruction, and occasionally may become very large. Hydatid and dermoid cysts are uncommon; they are easily identified.

Multilocular cysts. Multiple cysts are occasionally confined to one region of a kidney, which may resemble in size and appearance the cysts with polycystic disease. The cysts are compactly grouped and are usually situated in one pole of the kidney, while the remaining tissue is normal. The condition probably has its origin in tubular obstruction, although the possibility of its congenital nature is not excluded. According to our observation, this condition is unilateral. It is probable that it has been confused with polycystic disease in those cases in which polycystic kidney has been reported as being unilateral. Six cases of multilocular cysts have been observed at The Mayo Clinic.

Histology. Histological study of polycystic kidneys reveals the cysts to be lined usually with a low, flattened type of epithelium; occasionally proliferating areas are seen in which tuft-like papillary bunches collect, which often resemble glomeruli. The walls of the cysts are surrounded by a peculiar, pale-staining connective tissue, which fills the spaces between the cysts where the renal parenchyma has been completely destroyed. The content of the cysts is usually an albuminous material containing epithelial cells. Occasionally blood is found. Cholesterol crystal clefts and phagocytic cells containing fat particles are noted often. Depending on the degree of involvement, there may be areas of parenchyma, varying from those which appear to be fairly normal, to areas in which all parenchyma has been replaced by cystic degeneration. Numerous areas of localized lymphocytic infiltration, with hyalinization of glomeruli are present.

There is usually marked thickening of the smaller arteries and arteriolar walls. This is a very important factor in the progress of the disease, and may be closely associated with hypertension, which was present in a large number of our cases (7).

Groups of deeply staining, small round cells in a fetal kidney which is the site of congenital polycystic disease, have been interpreted as cells of an embryonic nature by Davis and others. In adults with arteriosclerotic changes in the kidney there are cells closely resembling these, which are probably lymphocytes.

HEREDITY

The hereditary tendency of polycystic kidney has been noted by many observers, and constitutes one of the chief proofs of its congenital nature. Numerous instances in which several members of a family, or members of a preceding and a succeeding generation were afflicted with polycystic disease were noted in our series of cases. In one case the condition was apparently present in representatives of four generations. In 2 cases it had been present in three generations, and in 5 cases in two generations. There is apparently variability in the frequency of inheritance in different families. In view of the pronounced hereditary tendency, it would seem logical to consider limitation of progeny. In some cases, sterilization may well be advocated.

AGE AND SEX

Clinical symptoms seldom appear in the first two decades of life and are unusual even in the third decade. If the lesion is not evident in infancy, it rarely manifests itself before puberty. Impairment of renal function

or other lesions are not usually evident until the fourth decade. The symptoms have their onset most frequently in the fourth or fifth decade. Eighty-eight patients, 46 per cent of our series, were included in this period of life. A large number of patients has no subjective evidence of disease until the sixth and seventh decades. The oldest patient observed in our series completed a normal span of life; namely, 69 years, before any symptoms appeared.

Several limited series of patients with polycystic disease have been reported in which the incidence was higher among males than among females. This preponderance of males was not borne out in our series of 193 patients; 98 were females, and 95, males.

DURATION OF DISEASE

Of 74 patients reported dead, 22 (approximately 30 per cent) died within 2 years following the onset of the first symptoms. Eleven (15 per cent) died in the period between 2 and 4 years after their first symptoms appeared. In these two groups 45 per cent, approximately half of the patients, lived less than 4 years following onset of their first symptoms. The remaining patients lived from 5 to 20 years, with the exception of 2, one of whom lived 23 years and the other, 36 years. The average age at death in this group was 50 years. Of the 42 patients reported living when last heard from, 25 had lived 10 years or more, and 9 had lived 20 years or more. From studies of the renal function, the cardiovascular system, the hæmoglobin, and the urine, one may obtain a fair idea of the expectancy of life of the patient.

The length of life will depend largely on the degree to which renal function is maintained, and this, in turn, on the degree of pressure exerted by the cysts on the normal renal tissue. There is marked variability in the incidence and size of cysts. When they are so numerous as to preclude much residual functioning renal tissue, and unless they remain very small, renal function will gradually diminish. This probably will depend on increase in size of the cysts rather than on the number of cysts. The prognosis for the patient who is in the third or fourth decade of life is uncertain, since evidence of subnormal renal function usually is not marked until the fifth decade. The patient observed in the fifth or sixth decade, who shows no evidence of diminished renal function, usually will live a life of normal length.

CLINICAL COURSE

The clinical picture presented by patients suffering from polycystic renal disease may vary considerably, depending on the stage to which the disease has progressed. The onset of symptoms may be very gradual, or extremely sudden. In cases of gradual onset the kidney may go on to almost complete destruction before any untoward symptoms are noted. As the cystic disease progresses, symptoms caused by renal insufficiency will appear, such as weakness, periods of malaise, headache, and gastric distress. Some patients suddenly give signs of renal insufficiency and uræmia, and may die in relatively short periods of time. Occasionally, cerebral hæmorrhage occurs in cases in which there is hypertension which had previously caused no distressing symptoms. Complications from intercurrent disease may appear as the result of decreased vitality and lowered values for hæmoglobin, and may add materially to the rapidity of the patient's decline. With increased vascular disturbance there may be symptoms caused by hypertension. With cardiac failure, which usually appears late, there may be œdema and dyspnœa.

Patients may be unaware of the gradual, progressive enlargement of the kidneys, which may have extended over many years prior to the onset of the first symptoms. Both kidneys may be found greatly enlarged, without apparent serious injury to renal function or to the cardiovascular system. After a varying period, however, the kidneys become insufficient, the cardiovascular system is impaired, and death will follow as a result of uræmia or vascular accident.

SUBJECTIVE CLINICAL DATA

Pain. Pain referred to the lumbar region, or to an upper, lateral abdominal region is a common symptom, and is often the reason for seeking medical aid. The pain is usually unilateral, and is described as a dull ache,

although it may become severe. It may be difficult to ascertain its cause, but in some cases in which there is more or less constant pain it is apparently the result of the excessive weight of the greatly enlarged kidney. In others it might be explained by intrarenal or intracystic pressure resulting from increase in size of individual cysts. Occasionally one or more cysts may become of enormous size and be the cause of considerable discomfort. Mechanical pressure on surrounding organs by these cysts may also be a factor. Complete relief has been obtained in several of our cases by surgical evacuation and destruction of large or hæmorrhagic cysts. Some patients obtained relief by lying down; others by supporting the kidneys with abdominal pads. Sudden hæmorrhage into a large cyst may be the cause of severe, acute pain. The passage of blood clots, with ureteral obstruction, may also be the cause of acute renal pain. Renal pain may be accompanied by fever and other evidence of acute renal infection, which may be explained by acute infection of one or more cysts, or by ascending pyelonephritis caused by temporary obstruction of a calyx.

Urinary symptoms. Gross hæmaturia was noted by 66 patients, approximately a third of the total number. It usually occurred at irregular intervals, was of limited duration, and was frequently brought on by violent exercise or jarring. As a rule the hæmorrhage was considerable and often accompanied by clot, which occasionally caused renal colic. The hæmaturia is similar to that occurring with renal neoplasm and if but one kidney is enlarged, the two conditions may be easily confused. Microscopic evidence of blood was reported to have been found in the urine of 85 patients. Dysuria or frequency of micturition was noted by only 15 per cent of patients.

Miscellaneous symptoms. Nausea and vomiting were noted in 50 cases, and were usually due to renal insufficiency. Loss of weight was noted in 115 cases (76 per cent). Weakness, usually profound, was present in 40 cases. Both loss of weight and weakness are probably the result of, and closely associated with, renal insufficiency. The physician should interpret the patient's report of weakness and of loss of weight of recent origin as usually presaging other evidence of renal insufficiency and as indicative of a serious prognosis.

OBJECTIVE CLINICAL DATA

Abdominal tumor. On physical examination, bilateral enlargement was palpable in 151 cases, unilateral enlargement in 30, and no enlargement in 12. Although both kidneys are usually markedly increased in size, there is often a decided difference in volume. It is not unusual to find one kidney but little larger than normal, while the other may be several times as large. Failure to determine bilateral renal enlargement on abdominal palpation is a common cause of error in diagnosis, and the lesion is frequently mistaken for renal neoplasm. Other factors, such as fat or muscular abdominal walls, may interfere with clinical recognition of renal enlargement of moderate degree. It is surprising how large the kidney may be and still escape detection in the general physical examination. As the kidneys become enlarged they frequently change their position. They usually assume a lower level and the mistaken diagnosis of simple renal ptosis often is made. Occasionally, lateral displacement of one or both kidneys occurs.

Although the tumor is often cystic, on abdominal palpation, particularly if there are several distended cysts on the anterior surface of the kidney, it may appear to be firm and solid. In some cases the irregularity of the cysts can be palpated easily. The kidneys move with respiration in many cases, while in others they become fixed as the result of perirenal adhesions. If, therefore, cystic irregularity and soft consistence are not present, but instead a unilateral, firm, fixed mass is palpated, as often occurs, the erroneous diagnosis of renal neoplasm is easily and frequently made.

In several cases varicocele of recent origin occurred on the left side, with marked enlargement of the left kidney, and would indicate pressure on the large veins on that side. This, also, might lead to confusion with renal neoplasm because of its frequent occurrence with this condition. Associated congenital deformities were found in several cases.

Blood pressure. Although one of us (Braasch) first called attention to the fre-

Fig. 1. Typical deformity with polycystic kidney, characterized by elongation of infundibula and irregular enlargement of minor calyces.

Fig. 2. Similar elongation of infundibula (shown in Figure 1), with crescent-shaped indentations of minor calyces caused by cysts.

quent occurrence of hypertension with polycystic kidney in 1916, it has since been a disputed point among clinicians. Bell and Clauson, in 1928, concluded that, "The available information is strongly against the view that congenital cystic disease of the kidneys is accompanied by persistent hypertension." This statement is not borne out by our present studies. The blood pressure of 190 patients was noted. Since the average age of our patients was 43 years, it would be logical to consider a systolic blood pressure of 145 millimeters of mercury or more, as an indication of hypertension; a diastolic pressure of 90 or more was similarly considered. Of our patients, 61 per cent had systolic pressures of 145 millimeters or more. In a study of the patients reported dead, records showed that 71 per cent had had elevated systolic pressure. In a control group of the same age and sex, made up of patients suffering from chronic pyelonephritis, 26 per cent had elevated blood pressure, and the incidence of hypertension among patients who were less than 50 years of age was 17 per cent. Among the patients with polycystic kidney who were reported dead, 52 per cent of those with hypertension were less than 50 years of age. Similarly, diastolic pressure was found to be more than 90 millimeters of mercury in 55 per cent of the cases, and more than 95 millimeters of mercury in 47 per cent. This indicates that the basis for hypertension in polycystic kidney is not directly related to age, but has its origin in vascular disease. This would be corroborated by the work of Hinman and Morrison (1924) and that of Ritter and Baehr (1928), who reported the results of injecting the arterial supply of polycystic kidneys. The latter two workers found a decrease in the size of the small arteries and the arterioles, and pointed out the similarity between the clinical course in cases of malignant hypertension and late glomerular nephritis with that of polycystic kidney.

Ocular fundi. There were abnormalities of the ocular fundi in 57 per cent of our cases.

Fig. 3. Semicircular indentation of calyces by cysts, with elongation of calyces and lateral displacement of pelvis.

In 20 per cent there were retinitis and associated retinal sclerosis. In 31 per cent there was retinal sclerosis unassociated with retinitis. In 6 per cent the patients had retinitis only, and without changes in the retinal vessels. In 43 per cent, examination of the ocular fundi gave negative results. It is evident, therefore, that the disease involves the entire vascular system.

Hæmoglobin and erythrocytes. Brown and Roth have pointed out that there is a definite relationship between the degree of renal insufficiency and the degree of anæmia. In their studies, the anæmia of chronic nephritis had definite prognostic value approximating that of retention of creatinine. In our group of patients with polycystic kidney the value for hæmoglobin of 93 (56 per cent) was 70 per cent (Dare) or less. Erythrocyte counts were less than 4,000,000 in 61 cases (42 per cent).

The urine. The specific gravity of the urine was less than 1.010 in 40.4 per cent of our cases. The value of low, fixed specific gravity from a prognostic standpoint is considerable. Albumin, usually in small amounts, was found in 180 cases. Urinary casts were observed in 21 cases (11 per cent). Microscopic evidence of hæmaturia was noted in 85 cases (43 per cent). Pus cells were found in 180 cases (91 per cent) in routine examinations of the urine. Catheterized urine from either kidney often contains a variable number of pus cells. This is apparently the result of inadequate intrapelvic drainage, caused by cysts compressing the pelvis or calyces. Ureteral catheterization occasionally will cause acute exacerbation of the infection, and should be avoided when possible. Materials injected in the course of retrograde pyelography may be retained in the pelvis and may be the cause of serious renal infection. Organic compounds of iodine which recently have come into use are less irritating, but even with these it is advisable to leave the ureteral catheter in place for several hours in order to drain the renal pelvis, and to perform lavage of the pelvis with sterile solutions of boric acid. Although in some specimens obtained by renal catheterization only occasional pus cells were found, this does not exclude previous renal infection, and cultures were in some cases positive.

Renal function. Regarding excretion of phenolsulphonephthalein of 40 per cent or less as indicative of renal insufficiency, renal function was subnormal in 67 per cent of our cases. In 26 cases, return of phenolsulphonephthalein was 31 to 40 per cent; in 21, 21 to 31 per cent; in 23, 11 to 20 per cent; in 18, 1 to 10 per cent; and in 27, only a trace of the dye, or none of it, was returned. In most cases there was delayed excretion of phenolsulphonephthalein as shown either by a retarded appearance time, or preponderance of excretion of dye in the last hour. Normal excretion of phenolsulphonephthalein should not be regarded as excluding polycystic disease, however. In many cases, although the specific gravity of the urine was found to be within normal limits (1.014 to 1.020), yet excretion of phenolsulphonephthalein was greatly reduced. Of interest is the lack of parallelism between the excretory and the retention tests for renal function in the earlier stages of renal insufficiency. Excretion of phenolsulphonephtha-

lein is often markedly reduced, whereas the value for blood urea is normal or only slightly elevated.

Blood urea. The value for blood urea was estimated in 117 cases of this group. In 78 cases (67 per cent) a value of 40 milligrams or more in each 100 cubic centimeters was noted; in 2 cases, it was more than 200 milligrams. In only 33 per cent was the value for blood urea normal. It is remarkable to what extent tolerance to renal insufficiency may be developed with a concentration of blood urea of more than 100 milligrams in each 100 cubic centimeters. A number of cases has been observed, in which the value for blood urea was 150 and 200, and the patients were in a fair degree of health for several years. This is also true when the value for creatinine is between 5 and 10 milligrams for each 100 cubic centimeters of blood.

Fig. 4. Abbreviation of calyx by cyst; rupture of wall of cyst, connecting it with pelvis.

UROGRAPHIC DIAGNOSIS

The recognition of polycystic kidney is frequently impossible without bilateral pyelography. The deformity of the pelvis as the result of polycystic disease is usually quite typical and easily recognized. It may, however, be unusual in outline and simulate deformity seen with renal neoplasm so closely that identification is impossible. In such cases a pyelogram of the opposite kidney is necessary. If that pelvis is shown to be normal, polycystic kidney can usually be excluded.

The deformity with polycystic kidney seen in the urogram has been described (3) and is characterized largely by marked elongation of the infundibula with crescent-shaped or bulbous enlargement of the minor calyces. With this there is frequently deformity and displacement of the renal pelvis laterally and downward. Occasionally there is obliteration of one or more of the calyces by pressure from cysts.

Bilateral simultaneous pyelography in cases of polycystic kidney was a dangerous procedure with the mediums formerly in use. With the employment of the organic compounds of iodine for retrograde pyelography this danger has been largely reduced, but even so one should hesitate to employ bilateral pyelography in the presence of polycystic kidney. The advent of intravenous urography has offered an ideal means of diagnosis in many cases. If the outline of the pelvis and calyces is clearly visualized, the condition can be recognized without much difficulty. Unfortunately, however, owing to delay in excretion resulting from renal insufficiency, the details of the pelves may not be clearly visualized. This is particularly true where the blood urea is more than 60 or 70 milligrams. Nevertheless, even in such cases, evidence of delayed excretion, fragmentary visualization of elongated calyces, and other data will offer a clue to the diagnosis. In cases of suspected polycystic kidney, therefore, intravenous urography should first be employed, and it frequently will obviate the necessity for cystoscopy.

TREATMENT

Knowing the condition present, much can be accomplished by means of general treatment and advice to the patient, who should be informed as to his condition and the usual prognosis. General directions as to the patient's regimen, such as dietary precautions consisting largely of restriction of protein, partaking of simple foods, and supervision of the amount of exercise, hours of rest, and

removal of causes of worry and stress are important factors. Urinary symptoms, as a result of secondary infection, are best controlled by general antiseptic measures rather than by local treatment.

It has been said that this condition is never surgical. However, experience has shown that surgical treatment is often of the greatest value to the patient. Although accidental removal of an uncomplicated polycystic kidney might well be disastrous if the function of the other kidney were insufficient, nevertheless, complications may exist which justify surgical treatment. Our experience in this field will be dealt with more fully in a subsequent contribution.

SUMMARY AND CONCLUSIONS

The average age of the patients at the onset of symptoms was 38.8 years. The average duration of life of the patients reported dead was 50 years. There was definite evidence of a hereditary trend. A systolic blood pressure of 145 millimeters of mercury or more was found in 61 per cent of the cases; the diastolic blood pressure was more than 90 millimeters in 55 per cent, and more than 95 millimeters in 47 per cent. Peripheral sclerosis was observed in 15.4 per cent. Retinal sclerosis, with other ocular changes, was noted in 51 per cent. Laboratory evidence of renal insufficiency was present in more than 60 per cent of the cases. Surgical complications occurred in approximately 30 per cent, which is a much greater incidence than is usually recorded. There is frequently a lack of parallelism between the retention and excretory tests for renal function; evidence of reduced renal function is usually greater in the latter.

The importance of vascular changes in cases of polycystic kidney has been underestimated in the past. Arteriosclerotic processes may be falsely attributed to developmental disturbances in the tubules and glomeruli. Although a developmental defect probably is primary in the etiology of congenital polycystic kidney, many of the clinical and pathologic manifestations have their origin in an altered condition of the vascular system.

Renal polycystic disease is easily overlooked, and usually is overlooked in the course of general clinical examination, since there are often no symptoms present which would indicate renal involvement. The renal origin of the patient's symptoms is often recognized only in the course of routine study of renal function. The condition may be confused with nephritis unless careful abdominal palpation is made. Failure to discover that renal enlargement is bilateral may lead to the erroneous diagnosis of renal neoplasm. Bilateral urographic studies may be necessary to determine involvement of both kidneys when abdominal palpation reveals unilateral enlargement. The most common symptom is a dull pain, usually referred to either renal region. Urinary symptoms of moderate frequency and dysuria are often observed. Gross hæmaturia occurs in approximately 33 per cent of cases and may simulate that occurring with neoplasm. The first clinical symptoms are frequently those of renal insufficiency, although a remarkable degree of tolerance is often noted in the presence of advanced renal destruction. Laboratory evidence of marked reduction in renal function, with comparatively few subjective symptoms, in the case of an adult who is in the third or fourth decade of life, should call attention to the possibility of polycystic renal disease. The prognosis will vary largely with the degree of renal dysfunction. If renal function remains normal, the prognosis is good. Even moderate reduction of renal function may remain stationary for as many as 10 or 15 years. When the reduction is advanced, the prognosis becomes grave, although several years may elapse before death. Expectancy of life will average almost 50 years, although patients are frequently observed who are more than 60 years of age. The hereditary nature of the disease should discourage the having of progeny, and sterilization should be considered.

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THE ANATOMY OF THE VEINS OF THE GALL BLADDER

Their Relation to an Impacted Stone

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From the Pathological Institute of the Saint George General Hospital in Hamburg, Prof. F. Wohlwill, Director; and the Pathological Institute of the German University of Prague, Prof. A. Ghon, Director

In his textbook of pathology Kaufmann makes the following statement in regard to the effect of obstruction of the cystic duct (usually produced by impaction of a stone): "Obstruction of the cystic duct usually produces hydrops of the gall bladder. If the obstruction is complete, no fresh bile can flow into the bladder, the bile in the bladder is absorbed by the lymph vessels of the mucosa, and the organ gradually becomes distended with a clear fluid produced by the mucosa. . . . If the obstruction is followed by infection of the gall bladder due to pyogenic organisms from the intestine, empyema of the gall bladder may develop, or a severe and destructive phlegmonous inflammatory process; for occlusion and retention raise the virulence of the bacteria. It is assumed that an empyema after healing can gradually change into a condition of hydrops." According to this prevailing conception, obstruction may lead to infection and infection produces inflammation.

In the study of a long series of extirpated gall bladders, Denton obtained little evidence of infection, and the anatomical and histological changes which he observed suggested interference with the circulation of the gall bladder, rather than an inflammatory process. In cases examined within 2 to 3 days after impaction of a stone in the cystic duct, he found in the wall of the gall bladder: œdema, venous distention, and hæmorrhage or hæmatoma. He attributed these circulatory changes, not to an acute inflammatory reaction, but to the pressure of the stone on the veins and lymphatics which drain the gall bladder. He stated that the veins and lymphatics are much more intimately incorporated in the cystic duct than is the cystic artery; and that impaction of a large stone in the cystic duct by direct pressure closes off the veins and lymphatics before the artery and causes varying degrees of circulatory interference.

The lesions produced by impaction of a stone fell into three groups: In 8 mild cases the gall bladder showed only venous distention and œdema. In several specimens analogous but more extensive lesions occurred, and there was widespread hæmorrhage. In the most severe cases the gall bladder showed many of the features of hæmorrhagic infarction.

Such a case with a similar interpretation was recently reported by Holden. At operation a stone measuring 3 by 4 by 4.5 centimeters was found tightly wedged in the neck. The gall bladder was extremely distended and filled with blood; the wall showed œdema, hæmorrhage, vascular distention, and gangrene of the fundus. These changes suggested that the venous return had been entirely shut off.

Denton holds that infection and inflammation, when they do occur, are secondary to mechanically produced circulatory changes.

The purpose of this study was to find out whether the anatomy of the veins of the gall bladder favored Denton's viewpoint.

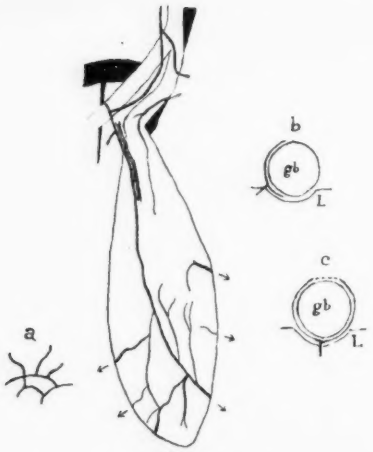

Fig. 1.

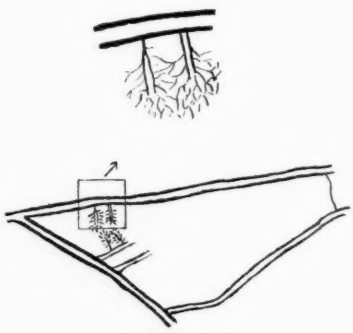

Fig. 3.

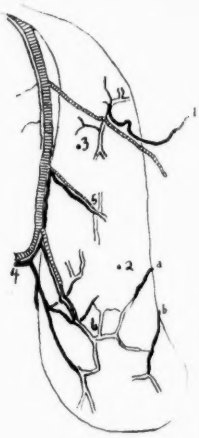

Fig. 4.

Fig. 2A.

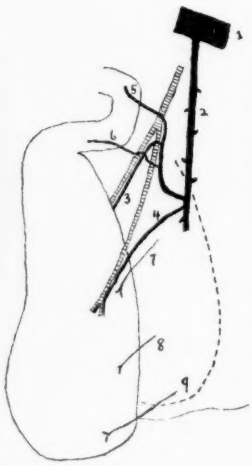

Fig. 2B.

Fig. 1. Normal gall bladder. The gall bladder is covered with a plexus of veins, portions of which are shown in the main sketch of the peritoneal surface and in the side sketch, a. From the plexus veins of various sizes lead directly into the liver (see arrows). Another vein leads from the plexus along the superficial branch of the cystic artery (cross-hatched), crosses the bile duct, and joins a small offshoot of the left branch of the portal vein. Side diagrams b and c represent cross sections of the gall bladder (*gb*) as it lies in the gall-bladder fossa of the liver (*L*). They show how the veins enter the liver. Some enter at the margin of the gall-bladder fossa, receiving tributaries from both peritoneal and deep surfaces (b). Veins may leave the plexus at this point or at any point deeper in the gall-bladder fossa. When a vein enters the liver at the bottom of the fossa (c), the arrangement resembles that of the veins draining the intestine.

Fig. 2A. Normal gall bladder. The photograph of the peritoneal surface was taken under water, after reflection of the serosa and brushing off the surface to remove loose connective tissue. The gall bladder is covered with a plexus of paired veins, between which run the uninjected arteries. Short anastomoses between paired veins are visible. Some of the small veins are unpaired. The smallest veins form a very fine plexus (Fig. 3), which, if completely injected, would probably fill the interstices of the coarser network of veins. The large veins, which converge upward and to the left, accompany the ramifications of the superficial branch of the cystic artery. The white spots were produced by rupture of veins and escape of injection fluid.

Fig. 2B. The sketch shows the lateral aspect and undersurface of the gall bladder of figure 2A, after it had been dissected free from its bed in the gall-bladder fossa (indicated by a broken line) and turned to the left. Only the arteries and veins running to and from the organ are drawn. The cystic artery (cross-hatched) comes down and divides in the usual manner into a superficial and deep branch; the superficial branch on the left runs around the bladder to supply the peritoneal surface; the deep branch is shown on the right, running down to supply the deep surface. The veins are numbered: 1 is the right main branch of the portal vein; 2 is a vein which communicates with 1, sends branches into the liver, all

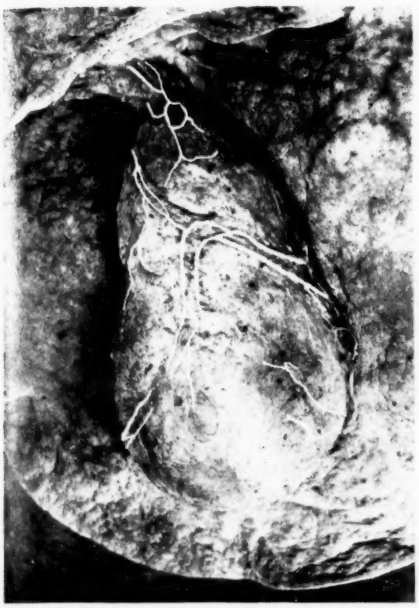

Fig. 5. Cholelithiasis, with a stone measuring 3.0 by 1.9 centimeters lodged in the upper portion of the body of the gall bladder. Distention of the organ below the stone. Injected veins traced in white.

Fig. 6. Cholelithiasis, with a stone measuring 2.4 by 1.7 centimeters impacted in the neck, and a much smaller stone in the cystic duct. A small vein running along the duct ends abruptly over the small stone, having apparently been obstructed by the stone.

LITERATURE

The circulation of the gall bladder was studied by Sappey, the famous French anatomist. His description of the veins is as follows:

"The veins fall into two groups: those which originate in the superior [peritoneal] surface of the gall bladder and those which originate in the inferior surface. The former usually give rise to two trunks which, either separately or after uniting, empty into the right branch of the portal vein. The latter, twelve to fifteen in number, representing so many small accessory portal veins, leave the

along its course, and, entering the gall-bladder fossa, receives *3* and *4*, which are the companion veins to the branches of the cystic artery. *3* receives two small veins, *5* and *6*, from the cystic duct. Veins *7*, *8*, and *9* drain the undersurface of the bladder and run upward into the gall-bladder fossa to enter the liver.

Fig. 3. Normal gall bladder. The injection revealed a plexus of paired veins similar to that shown in Figure 2A. The upper sketch shows on a magnified scale the partially injected fine plexus. The blood from this plexus is taken up by forked and paired veins and carried to one of two larger paired veins. The source of the field of the upper sketch is shown in the lower sketch, in which a portion of the plexus of large paired veins is portrayed.

Fig. 4. Normal gall bladder. The sketch shows the peritoneal surface. An artery (cross-hatched) runs along the medial side of the gall bladder in the fissure between gall bladder and liver, giving off small branches to the peritoneal and deep surfaces of the gall bladder. Three of these branches are shown in the sketch. The uppermost crosses the gall bladder and runs on into the liver. The two lower ones sink gradually into the plane of the paired venæ concomitantes, which cover the gall bladder, just as in Figure 2A. These paired veins are drawn in only at the points of origin of the veins draining the gall bladder. A vein accompanies for a short distance the uppermost branch of the large artery and enters the liver at *1*. Veins *a* and *b* unite under the gall bladder to form a vein which runs into the liver at *2*, not far from the bottom of the gall-bladder fossa. Another vein enters the liver under the gall bladder at *3*, collecting blood from the cranial portion of the deep surface. The large vein *4*, which enters the liver with the large artery, receives three tributaries from the peritoneal surface and one from the deep surface. The sketch shows how large unpaired veins arise from the smaller paired veins of the cystic plexus. At their point of origin they leave the plane of the paired veins and become more superficial.

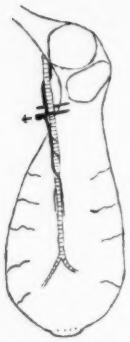

Fig. 7.

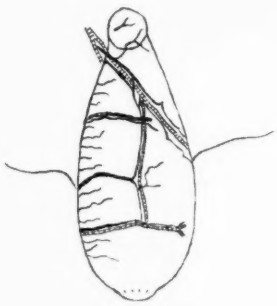

Fig. 8A.

Fig. 8B.

Fig. 9A.

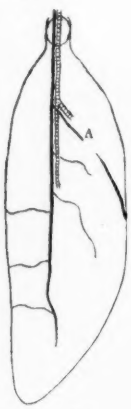

Fig. 9B.

Fig. 7. Cholelithiasis, with a rounded stone, 1.6 centimeters in diameter, firmly impacted in the neck. Below this stone lies loosely another somewhat smaller stone, which can be readily pushed toward the fundus. There is a small herniation of the wall at the fundus. The arrow indicates the point where a large vein enters the liver.

Fig. 8. Cholelithiasis, with a rounded stone, 1.75 centimeters in diameter, impacted in the neck. There is a herniation at the tip of the fundus. The usual injection of the cystic veins was followed by an India ink injection of the cystic artery (cross-hatched). Figure 8A shows the medial half of the peritoneal surface; Figure 8B, part of the lateral half of the peritoneal suface.

Fig. 9. Cholelithiasis, with a small stone impacted in the first portion of the cystic duct. Hydrops. The photograph (Fig. 9A) of the uninjected specimen shows the peritoneal surface of the gall bladder as seen from the right. The veins are filled with blood resulting in a remarkably complete "natural injection." Nearly all of them lie at right angles to the long axis of the bladder, anastomose freely, and run directly into the liver at the margin of the gall-bladder fossa. Along the lower margin of the "high light" runs a large vein (partly hidden), which follows the course of the superficial branch of the cystic artery and terminates at *A* in Figure 9B. Figure 9B shows the opposite side of the gall bladder.

gall bladder to ramify in the liver lobules which surround the gall-bladder fossa."

My own study of the anatomy of the veins, both in the normal gall bladder and in gall bladders with a stone impacted in the [illegible]ck or cystic duct, confirm observations of Sa[illegible]ey in general, if not in particular, and show th[illegible]n impacted stone cannot produce venous st[illegible]is.

Fig. 10.

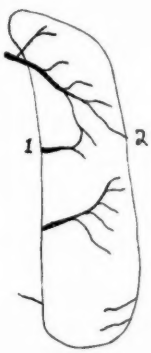

Fig. 11A.

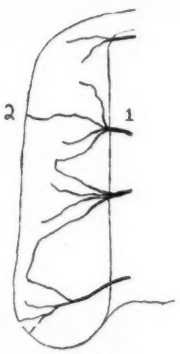

Fig. 11B.

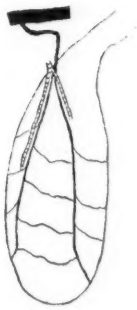

Fig. 12.

Fig. 10. Cholelithiasis, with a stone the size of a cherry impacted in the cystic duct. Gall-bladder wall shrunken and indurated. Veins run in three directions from the stone in the cystic duct. The fundus of the gall bladder is drained by numerous veins, not drawn in the sketch.

Fig. 11. Cholelithiasis without impaction. The wall is thickened in its middle third. Figure 11A shows the medial side of the gall bladder; Figure 11B, the lateral aspect of the bladder. The large trunk, marked *1* in Figure 11A, is shown again in Figure 11B. An anastomosis across the top of the bladder is marked *2* in the two figures.

Fig. 12. Cholelithiasis without impaction. A diagrammatic sketch of the peritoneal surface. The two large trunks unite to form a single vein which runs into a large branch of the portal vein.

TECHNIQUE

A method of injecting the veins of the gall bladder was outlined for me by Dr. Blotevogel of the Anatomical Institute of the University of Hamburg. In order to obtain a complete injection it was necessary to inject not only the gall bladder, but also all the surrounding organs. A description of the perfected technique follows:

After opening the body cavities sever the small intestine at the junction of the duodenum and jejunum and remove the jejunum, ileum, and colon. Clamp the inferior vena cava close to the right auricle and sever it above the clamp. Remove the heart and lungs. Remove the diaphragm and the remainder of the abdominal organs in one piece. To avoid tearing the liver capsule, pull the organs straight forward. Take care not to cut into the liver capsule. Separate the kidneys and the rest of the urogenital organs, leaving the suprarenal glands in place. There now remain in one piece the diaphragm, liver, stomach, duodenum, pancreas, spleen, suprarenals, abdominal aorta, and inferior vena cava. Tie off the inferior vena cava above the diaphragm and remove the clamp. Dissect out the portion of the vena cava below the liver and tie it off close to the liver. Following up one of the large mesenteric veins, find the beginning of the portal vein, and insert here a cannula plugged with cork (to prevent the entrance of water before the time of injection). Using one or more large curved or straight clamps, such as are used in operating rooms, clamp off as much as possible of the stomach, but especially that portion of the greater curvature affected by postmortem digestion. Place the group of organs in a bucket of hot water at 65 degrees (centigrade) for at least 15 to 30 minutes, or until the injection fluid is ready. The water should at the time of injection have a temperature of at least 40 degrees.

Place 250 grams of French gelatin in 1000 cubic centimeters of water for 1 hour, then heat over a water bath slowly to about 75 degrees. It is not necessary to filter at this point. After the gelatin has dissolved, add 50 grams of cinnabar (vermillion) or 40 grams of zinc white (preferable) and continue the heating and stirring until a homogeneous red or white fluid is obtained. Allow to cool to 65 to 70 degrees. If the liver to be injected is large, use 50 per cent more gelatin, water, and coloring matter.

Stretch two layers of gauze over a large glass funnel held in a ring stand. To the funnel attach a long rubber tube of large caliber, and clamp the lower end of the tube.

Remove the organs from the hot water and place them on an autopsy table. Pour the injection fluid into the funnel and, after it has replaced the air in the tube, remove the cork from the cannula and attach the tube. In order to carry out the injection very slowly, the funnel is held at the level of the organs at first and then gradually raised until sufficient pressure is obtained, but not so much as to burst the veins. The injection should last about one-half hour. The leaks in the preparation are washed off with running water and clamped. As soon as all leaks are stopped, the organs are replaced in warm water, without interrupting the injection.

After the injection, the preparation is placed in ice water in order to hasten the coagulation of the gelatin. When coagulation is complete, the cannula and clamps may be removed.

Examination of the liver after the injection shows that the portal vein and its branches contain the gelatin suspension of cinnabar or zinc white, while the hepatic vein and its branches contain gelatin mixed with blood, the liver capillaries having filtered out the coloring matter. This can be confirmed microscopically by making frozen sections. In the gall bladder the veins contain the injected pigment, while the cystic arteries contain gelatin mixed with blood. This interferes with a subsequent injection of the cystic artery and its branches. It also explains the failure of an attempt at simultaneous injection of the arteries and veins by way of the portal vein and cœliac axis.

In order to study the injected veins, incise the serosa of the gall bladder down the middle of the free surface and reflect it to either side. In so doing the small veins of the serosa are seen. Next reflect the layer of loose connective tissue in which course the artery and the veins which run to and from the gall bladder (cystic artery and veins). To complete this stage of the dissection it is necessary to lift the gall bladder from its bed. This lays bare the veins of the gall-bladder wall (cystic plexus). Open the gall bladder to inspect the veins of the mucosa (plexus of the mucosa).

A few injections of the cystic arteries, with a needle and syringe filled with India ink or Teichman's mass, were made for me by Professor Grosser, of the German University of Prague.

INJECTION OF THE VEINS OF THE NORMAL GALL BLADDER

The results of the injections of the normal gall bladder are shown in Figures 1 to 4. Preparations 2, 3, and 4 (Figures 2A, 3, and 4) show that the gall bladder is covered with a plexus of paired veins which accompany the branches of the cystic artery. This might be called the "cystic plexus" or "cystic plexus proper." Two subsequent preparations showed the same kind of a plexus, making a total of five successive injections in which the plexus of paired veins was constant. In Preparation 1, however, a plexus of unpaired veins was recorded (Figure 1 and side sketch a). Figure 3 shows a portion of the plexus of very fine unpaired veins which fills the interstices of the coarser plexus.

The "cystic plexus" is drained by a number of unpaired "cystic veins" which are very irregular in number, size, and course. Examples of them are shown in Figures 1, 2B, and 4 and in all of the subsequent figures (cases of cholelithiasis). They are especially clear in Figures 11A and 11B. Figure 4 shows how these large unpaired cystic veins arise from the smaller paired veins of the cystic plexus. Figure 2B shows how some of the cystic veins (*3* and *4*) accompany the main branches of the cystic artery, while others (*7*, *8*, and *9*) do not. Some of the cystic veins could be followed into large branches of the portal vein (Figures 1 and 2B); the others entered the liver and could not be followed by ordinary methods of dissection. They enter the liver at any point in the gall-bladder fossa from its margin (Figure 1, side diagram b) to its deepest point (Figure 1, side diagram c).

Not shown in the Figures are the plexus of the gall-bladder mucosa and the very small veins of the serosa. The plexus of the mucosa resembles the plexus of the duodenal mucosa, which is also injected in these preparations.

It consists of a rich network of small veins, the interstices of which are filled with a network of extremely small veins. Frozen sections of the wall of the injected gall bladder (after embedding in gelatin) show the veins of this plexus and also the veins of the cystic plexus proper, which lie outside the muscularis. The muscularis itself contains small veins, some of which unite the mucosal plexus with the cystic plexus.

The serosal veins in the various preparations take in general a lateral course toward the sides of the gall bladder. Some of them, and especially those in the middle of the peritoneal surface, communicate with the veins of the cystic plexus. The others run off to one side or the other into the liver.

INJECTION OF THE VEINS IN CASES OF CHOLELITHIASIS

The cystic veins were injected in 8 cases of cholelithiasis. In all of the cases the pathological condition of the gall bladder was a collateral finding at postmortem examination, death being due to other causes. In the first six cases there was an impacted stone. In most of these cases the bladder was hydropic and showed signs of a healed process as evidenced by scars in the mucosa, fibrous thickening of the wall with or without shrinkage, or adhesions of the peritoneal surface. The results of the injection in the various preparations are shown by photograph, drawing, or diagram (Figs. 5 to 12).

In Preparation 5 (Fig. 5) an unusually large stone was lodged in the upper portion of the body of the gall bladder. In this position the stone could conceivably press on the large veins which accompany the superficial branch of the cystic artery (uninjected) in its course from above on the left diagonally down across the bladder to the right. But from any hypothetical point of pressure the blood would drain off in at least two directions: up and to the left, or down and to the right by the two large veins which run directly into the liver.

In Preparations 6, 7, 8, 9, and 10 (Figures 6, 7, 8, 9, and 10), one or two stones were impacted in the neck or in the cystic duct. In the figures the injected veins are traced in black, while the visible portions of the arteries are cross-hatched. It was found that the veins in the region of the neck leave the gall bladder below the point where the stone was impacted; and, as the veins in their further course lie in loose connective tissue apart from the neck and cystic duct, they could not be pressed upon by the impacted stone. (In Figure 9B the artery and vein run past the stone impacted in the duct, but not close to it, as the figure would indicate.) Furthermore, there are so many veins running from all parts of the body of the gall bladder into the liver that it is impossible for an impacted stone to produce venous stasis.

The following experiment proves that, even if the veins at the neck of the gall bladder are occluded, adequate drainage would occur through the veins which run from the body of the organ directly into the liver:

In a case with a normal gall bladder, the neck of the gall bladder was freed from its attachment to the liver sufficiently to permit a ligature to be placed around it. With the ligature firmly tied, the usual injection through the portal vein was made. The injection of the cystic veins was as complete, above and below the tie, as if no ligature had been placed. From one side of the gall bladder four, from the other side three veins, ran directly into the liver. The injection fluid must have entered the gall bladder through these veins, because the veins at the neck were closed by the ligature.

The injury produced by cholecystitis destructiva could conceivably obliterate some of the veins of the gall bladder. Preparation 11 (Figures 11A and 11B) is of interest in this connection; for, although the gall bladder was thickened in its middle third, the even distribution of the veins was undisturbed.

The completeness of injection varied in the different cases of cholelithiasis, and was in no case as perfect as that attained in the injections of the normal gall bladder. The "natural injection" of the veins in Figure 9A is of special anatomical interest, because it was unusually complete and because the veins are not exaggerated in size, as in artificial injections. In Preparation 12 (Fig. 12) the course of the veins on the peritoneal surface agrees with the description of Sappey.

OBSTRUCTION OF THE LYMPHATICS

The lymphatics of the gall bladder were not studied. Sappey states that the lymphatics of the gall bladder and the adjacent parts of the liver converge toward and flow into a lymph node located at the neck of the gall bladder. A hypothetical obstruction at this point would in my opinion be relieved by drainage through the lymphatics of the liver; for the lymphatic system as a whole is characterized by an extraordinary richness in anastomoses, not only of the smaller but also of the larger vessels (Bartels).

SUMMARY

1. It has been maintained by Denton that impaction of a large stone in the cystic duct by direct pressure closes off the veins and lymphatics which drain the gall bladder and causes circulatory stasis, varying from simple distention to hæmorrhagic infarction. The anatomy of the veins of the gall bladder was studied to determine whether it supported Denton's viewpoint.

2. Injection of the veins of the normal gall bladder revealed a "venous plexus of the mucosa" similar to that of the duodenal mucosa; a "cystic plexus," which lies just outside the muscularis and usually consists of paired venæ concomitantes accompanying the ramifications of the cystic artery; and unpaired "cystic veins" (already described by Sappey as "accessory portal veins"), which drain the gall bladder. These cystic veins vary greatly in number, size, and course. Some of them accompany the branches of the cystic artery toward the neck of the gall bladder. Others carry blood around the sides of the gall bladder, or from its deep surface directly into the liver by way of the gall-bladder fossa.

3. Injections of the veins in cases of cholelithiasis with impaction showed that the impacted stone cannot by direct pressure cause venous stasis. This is impossible because:

a. None of the cystic veins run close enough to the cystic duct to be affected by the pressure of a stone in the duct. Only the veins of the cystic duct itself are affected by such a stone.

b. The cystic artery and its accompanying vein meet (or leave) the gall bladder below the point of impaction of a stone in the neck.

c. Only a very large stone lodged in the upper portion of the body of the gall bladder could press upon large cystic veins. In this case, on account of the rich anastomoses, blood would be carried away in at least two directions from the point of pressure.

d. Only a small fraction of the venous drainage occurs by way of the neck.

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THE RÔLE OF THE EXTERNAL SECRETION OF THE PANCREAS IN EXPERIMENTAL HIGH INTESTINAL OBSTRUCTION

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IT has long been known that high intestinal obstruction in dogs is compatible with only a short duration of life. Although there is a minor variation in the longevity of animals with obstructions just below the ligament of Treitz, the results of numerous investigators are substantially the same. According to Haden and Orr (7), who produced obstructions of the upper jejunum in 35 dogs, the average duration of life was 6.8 days. Wangensteen (14) found the length of life of the dog with high intestinal obstruction to be 3 to 4 days; while Dragstedt and Moorhead (1), who obstructed the duodenum below the pancreatic duct, observed that all animals died in less than 96 hours. The earlier studies of Hartwell and Hoguet (9) indicate that few dogs with obstructions 10 to 30 centimeters below the pylorus lived longer than 5 days.

J. W. Draper Maury (12), in 1909, drained the biliary and pancreatic liquids below the site of obstruction. The bile was found to be in no way connected with what he has termed a physiological death. Dogs in which the external secretion of the pancreas was drained into the bowel below the obstruction lived, whereas those in which it was drained above died. To produce drainage below the obstruction, the upper pancreatic duct was ligated and the duodenum obstructed between the ampulla of Vater and the major pancreatic duct. Again, Eisberg and Draper, in 1918 (4), drained the duodenum and its appendages below the site of upper intestinal obstruction. The entire duodenum with its outbuds, the pancreas and liver, were first separated from the alimentary tract. The pyloric end of the duodenal segment and the stomach were occluded, the duodenum was anastomosed to the jejunum, and posterior gastro-enterostomy was performed. This constituted the primary operation. Two to 3 weeks later the bowel was obstructed by section and infolding 35 centimeters aboral to the gastro-enterostomy. These animals lived 17 days, in comparison with 6 days in the control animals. They concluded that the duodenum with its appendages was responsible for death in high obstruction. In a later article Eisberg (3) excluded the bile from the obstructed segments, confirming the earlier work of J. W. Draper Maury. According to Sweet, Peet, and Hendrix (13), if the pancreatic ducts are ligated and a high obstruction produced, dogs will live somewhat longer than the average, but life is not strikingly prolonged. This same observation has also been made by Eisberg. This author also concluded that the pancreas is the main source of the toxin in duodenal obstruction. The work of Dragstedt et al (2) and Mann and Kawamura has shown that the entire duodenum may be surgically removed in animals without causing death if the bile and pancreatic secretions are preserved by transplanting the ducts of the gall bladder and pancreas.

In our review of the literature we find much experimental evidence pointing to the importance of the external pancreatic secretion as a lethal factor in intestinal obstruction. In order to gain further information in regard to this important secretion and its relation to intestinal obstruction, we have made a study in which the major portion of the external secretion of the pancreas is preserved by draining it into the jejunum below the point of the high obstruction. This is accomplished by transplanting a small section of the duodenum, containing the ductus Santorini, into the jejunum below the point of contemplated obstruction. Thus, the adjacent organs, namely, the stomach, liver, and the duodenum remain physiologically intact while the pancreas is drained into the unobstructed intestine. We have thus isolated only one organ, the pancreas, so that the preservation of the major portion of its

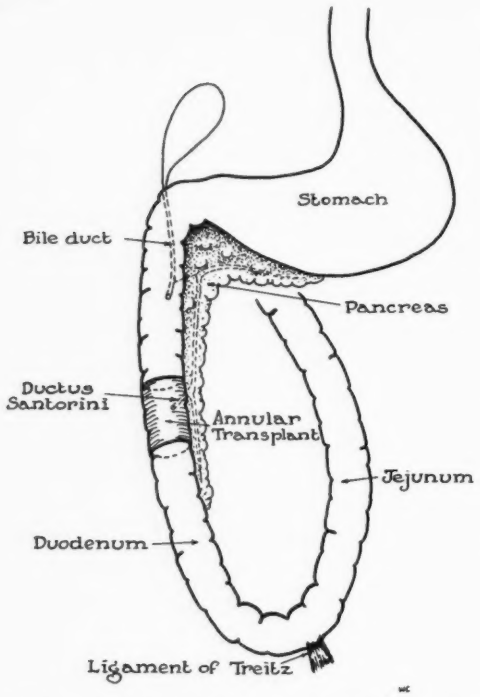

Fig. 1. Diagram showing section of duodenum with major pancreatic duct to be transplanted.

external secretion can be properly evaluated. These animals have received absolutely no treatment. Following the operations under ether anæsthesia they have been returned to metabolism cages and have been offered water *ad libitum*.

In dogs there are two pancreatic ducts. The upper, or duct of Wirsung, enters the duodenum at the ampulla of Vater with the gall duct. This duct is quite small and relatively of much less importance than the duct of Santorini. The latter duct conducts the major part of the secretion of the pancreas into the alimentary canal.

TABLE I.—BLOOD CHEMICAL STUDIES

After Obstruction of Jejunum Below Ligament of Treitz With Previously Transplanted Pancreatic Duct (D. Santorini) Distal to Obstruction

Dog No.	Day after operation (obstruction)	Blood				CO_2 combining power. Volume per cent	Weight in kilos
		Milligrams per 100 c.cm.					
		Urea nitrogen	Total non-protein nitrogen	Chlorides	Sugar		
1	0	12.6	32.1	500	84.8	40.4	6.95
	2	13.4	38.2	465	69.9	41.2	
	5	30.4	68.8	370	81.2	64.1	5.7
	9	40.6	71.4	340	93.0	69.7	
	12	15.4	35.8	325	103.5	83.4	4.8
	16	35.1	87.5	285	132.6	72.8	
	18	58.5	101.8	285	134.2	82.8	4.1
	19	Death					
2	0	7.7	32.8	538	67.2	47.5	13.6
	3	8.8	36.7	505	72.9	56.8	
	7	13.2	42.0	432	66.9	61.2	12.3
	12	10.7	47.1	315	75.7	84.4	
	18	9.7	36.6	325	87.4	82.8	
	22	8.8	44.1	340	76.6	83.5	11.8
	26	11.4	40.6	300	90.0	66.3	
	29	11.2	46.9	250	107.6	84.7	
	30	50.4	111.1	200	176.0	97.5	9.8
	31	Death					
3	0	11.1	31.4	469	69.0	44.5	8.7
	2	8.4	32.8	420	64.4	51.0	
	6	21.9	30.7	305	79.0	67.3	
	10	15.4	38.9	300	74.0	80.5	
	13	30.0	46.0	286	80.5	86.0	
	16	38.7	52.8	325	90.0	89.0	
	18	39.7	60.0	336	91.0	89.0	5.9
	19	Death					
4	0	9.8	33.7	470	64.4	40.4	12.7
	2	17.2	41.0	410	69.0	51.1	
	5	17.9	30.2	340	53.6	36.9	
	9	19.1	38.7	270	70.0	79.6	
	12	12.6	36.4	270	65.7	87.5	
	16	15.4	26.9	300	68.5	87.5	
	21	16.8	32.5	330	72.0	74.9	
	26	19.1	32.6	300	84.5	75.4	
	28	30.8	38.0	285	97.5	82.9	
	29	54.6	60.8	260	129.0	91.3	
	30	50.6	65.2	250	99.0	91.5	
	31	51.9	69.7	330	91.0	90.0	7.5
	31	Death					
5	0	8.8	27.3	480	64.3	41.6	7.4
	2	12.4	25.7	490	71.5	50.4	
	7	14.9	29.4	440	78.5	58.8	
	10	11.6	33.9	420	130.0	60.7	
	13	15.8	21.4	410	93.6	62.5	
	15	13.5	30.5	405	79.0	72.7	
	17	22.4	34.9	370	83.5	74.9	
	18	69.3	83.3	330	33.4	66.4	5.9
	18	Death					
6	0	20.0	40.6	490	60.0	42.6	7.7
	2	17.7	29.4	430	68.5	54.1	
	6	14.9	25.2	460	64.7	53.2	7.1
	13	19.1	35.7	360	91.0	71.8	
	17	27.5	45.0	360	87.0	83.4	
	21	50.9	45.6	320	132.0	71.8	
	22	79.6	131.5	310	134.0	60.1	5.1
	23	Death					

TECHNIQUE

In all, 6 animals are reported. The experiment was done in two stages. The first stage consisted in the transplantation of a section of the duodenum, approximately 3 centimeters in length, containing the duct of Santorini, into the jejunum 20 to 25 centimeters below the ligament of Treitz. This was accomplished by a double end-to-end anastomosis (Figs. 1 and 2). The continuity of the duodenum was established by an end-to-end union.

Following the above operation the animals were allowed to recover completely from the operation and regain any lost weight. The jejunum was then obstructed just below the ligament of Treitz above the transplanted pancreatic duct.

OBSERVATIONS

Chemical studies of the blood have been made at repeated intervals of each of the animals. The results are similar to those found in simple high obstruction of the jejunum (7), although the changes are slower in developing (Table I). A careful autopsy has been done on each dog and all animals excluded in which a patent ductus Santorini could not be demonstrated.

The recovery of the dogs was rapid after the second operation. They vomited infrequently and the loss of weight was quite gradual. Their period of survival was from 18 to 31 days, with an average of 23⅓ days.

At autopsy the animals showed a striking degree of emaciation. The panniculus adiposus had practically disappeared and the omentum was reduced to spiderweb consistency. There was practically no body fat to be found and the tissues possessed a waxy appearance. The intestine below the obstruction was thin and collapsed. Above the obstruction, the gut was uniformly enormously distended with liquid. There was always considerable liquid in the stomach. Both pancreatic and bile ducts were demonstrated as patent by the passage of a probe.

SUMMARY

When the major part of the external secretion of the pancreas was preserved by drainage into the intestine below the site of an obstructed jejunum the survival period of dogs in our series was 300 to 400 per cent longer than when the secretion was not preserved. The average duration of life of 6 dogs was 23⅓ days. These results closely parallel the published work of Eisberg and Draper (4) and of Jenkins (10), in which the secretions of the liver, duodenum, and pancreas are drained into the jejunum below the site of the obstruction. It is well established that if the first loop of the jejunum be divided and its proximal end drained to the outside, so that the upper intestinal secretions are lost, dogs die as quickly as if the jejunum had been simply obstructed (8).

It is now recognized that the pancreatic fluid is essential to life. Elman and McCaughan (5) and Hartmann (6) have demonstrated that the total loss of the external secretion of the pancreas in dogs results in death within 6 to 8 days. This is a comparable length of time to their survival period when the first loop of the jejunum is obstructed or completely drained.

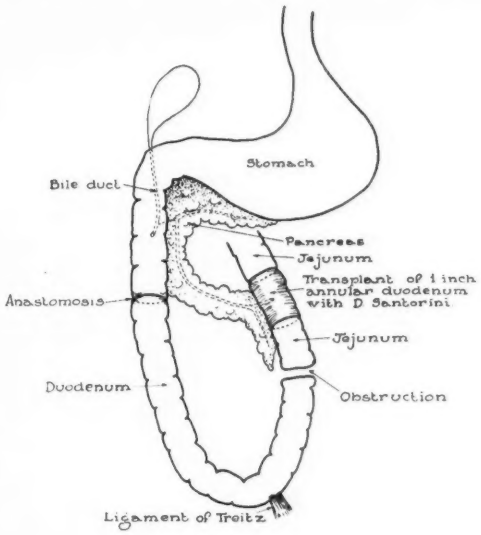

Fig. 2. Diagram showing transplanted section of duodenum with major pancreatic duct. The site of anastomosis and jejunal obstruction are also indicated.

CONCLUSIONS

1. A series of 6 dogs are here reported, in which the major pancreatic duct has been transplanted into the jejunum, and the pancreatic secretion preserved below a high jejunal obstruction. Such animals have lived an average of 23⅓ days.

2. Dogs with high jejunal obstruction, in which the external secretion of the pancreas was preserved, lived approximately three times as long as dogs having simple obstruction in the same location.

3. It seems quite evident from these experiments and those of others, that the early death from high intestinal obstruction is intimately associated with a loss of the pancreatic juice.

4. We do not attempt to explain what element or elements of the pancreatic juice, when preserved, tends to prolong life in acute high intestinal obstruction.

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INTRA-ABDOMINAL PRESSURES CREATED BY VOLUNTARY MUSCULAR EFFORT

I. Technique of Measurement by Vaginal Balloon

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MEASUREMENT of the maximum intra-abdominal pressure which can be created by voluntary muscular effort is of interest to the obstetrician and gynecologist in view of the rôle which it may play in relation to labor and to the etiology of prolapse.

Measurements can be made indirectly by recording pressure transmitted to an air inflated balloon within the vagina. The present report describes such a technique, discusses the relation between the vaginal and the intra-abdominal pressures, and records some measurements made by this method.

LITERATURE

Since the observations of Weber (1851), who first observed that extreme expiratory effort may diminish or obliterate the peripheral pulse, about 100 reports have been published which deal with intra-abdominal pressure in man. In general, these studies have followed three lines: (a) the relation of abdominal to atmospheric pressure, (b) the levels of pressure in different parts of the abdomen when at rest, and (c) the influence of respiration upon the pressure.

Experimental data are presented in about 50 of these reports of which 27 (Table I) record the pressure in man. Only three record pressures created by *voluntary muscular effort.*

MATERIALS AND METHODS

The technique outlined in the present study was developed upon 29 applicants to the Gynecologic Out-Patient Department of the Hospital of the University of Pennsylvania, and the measurements in Table II made on healthy volunteers.

Apparatus. The apparatus (Fig. 1) consisted essentially of a rubber balloon, which was an ordinary commercial condom, connected to a simple U-tube mercury manometer. The balloon was fastened by elastic bands to a perforated rubber stopper, which possessed a groove near one end and was fitted over a piece of brass tubing 9.0 centimeters long, with an outside diameter of 0.8 centimeter. An oval, metal shield, 9.0 centimeters by 5.0 centimeters was attached at right angles to the tube, 6.0 centimeters from its vaginal end. The apparatus was immobilized in the vagina by straps (2 anterior and 2 posterior) extending from the shield to a belt at the waist. The balloon was immobilized by a rubber cup fitted to the tube just below the rubber stopper. This prevented it from being forced down around its point of attachment and out of the vagina when the patient strained.

The cup (Fig. 2) was made of fine quality soft rubber. Its base was 2.5 centimeters in diameter, and 0.8 centimeter in thickness. Its sides were 5.5 centimeters high and 0.2 centimeter thick at the base, tapering to paper thinness at the rim, which was 8.0 centimeters in diameter. Insertion of the balloon and cup into the vaginas of deflorated patients was easy and the apparatus caused no discomfort while in position.

Technique of measurement. The bladder was emptied prior to each series of tests. The cup and balloon were placed in the vagina, the shield being placed firmly against the vulva, after which the straps were fastened to the belt at the waist. The balloon was inflated with air in order to fill the vagina, and the pressure within the system was regulated by movement of water in a pair of gravity bottles.

The patient was given preliminary instructions to strain slowly with maximum effort for each test. The tests were made at intervals of not less than 2 minutes, which we thought was sufficient time to afford an adequate rest period.

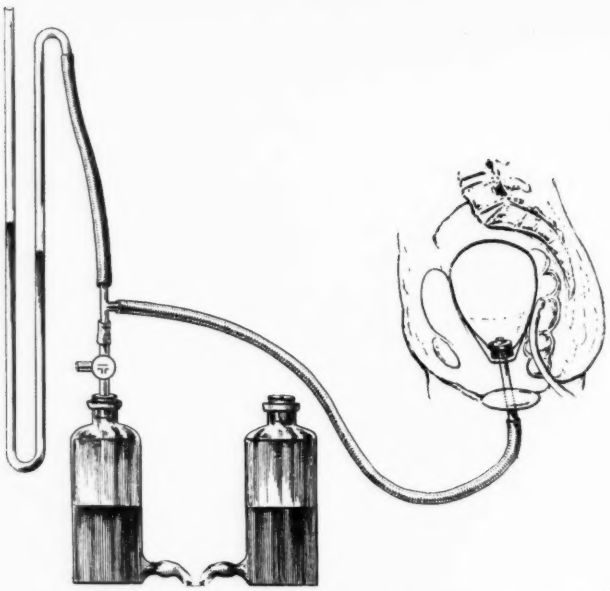

Fig. 1. Diagram of apparatus. Note balloon in vagina. In practice, the system contained air though in order to obtain the lateral roentgenogram from which this illustration of the pelvis was made, the vaginal balloon was filled with a roentgen opaque fluid. The balloon and vaginal apparatus are connected with a mercury manometer; the pressure within the system is regulated by movement of water in the gravity bottles.

The pressure recorded in the vagina when the patient strained was influenced by a number of circumstances:

1. Sudden, sharp, muscular contraction set the mercury column into simple rhythmic vibration. Under such conditions, the column of mercury reached a much higher level on the first up swing than when the muscular contraction was slow. However, if the patient was instructed to strain slowly and to maintain her maximum effort for an appreciable time, this difficulty was eliminated. These observations were confirmed by kymographic records of each type of muscular effort. They demonstrated clearly that sudden, sharp contractions gave erroneous results, but that slow, steady effort gave true readings.

Fig. 2. Detailed diagram of vaginal apparatus. Hollow metal tube with metal vulvar shield supports a partially inflated rubber balloon attached to rubber stopper, which is cemented to floor of a rubber cup.

2. The efforts of a patient on the first test day were usually weaker than subsequent ones, due to the newness of the experience and lack of habit formation.

3. The ability of the patient to equal her highest record on a given day, when under otherwise identical experimental conditions, was lowered by worries not connected with the test.

4. It was found essential to indicate the moment for each effort by a command, constant in word and tone. Deviation from this

TABLE I.—INTRA-ABDOMINAL PRESSURE IN MAN

Chronological table of reports in the literature which deal with the measurement of intra-abdominal pressure, and in which man was used as test object. Note: (1) that only 3 authors (Braune, 1865; Dubois, 1876; Hoermann, 1905) measured pressure on voluntary muscular effort; (2) that only two observers (Mathes, 1906; Bohnen, 1931) employed a balloon in the vagina; (3) that neither of the latter measured pressures created by voluntary muscular effort.

Date	Author	Method	Number of subjects	Voluntary effort
1865	Braune, W.	Tube in rectum	*	Measured
1872	Schatz, F.	Balloon in uterus during labor. Tube in stomach. Cannula in abdomen of cadavers	*	N.M.†
1874	Emminghaus, H.	Tube and balloon in œsophagus and stomach	*	N.M.
1875	Odebrecht, E.	Catheter in bladder. Tube in rectum	*	N.M.
1876	Wendt, E.	Tube in rectum	1 (self)	N.M.
1876	Dubois, P.	Balloon in bladder and rectum. Cannula in abdomen of patient with ascites	39	Measured
1878	Quincke, H.	Cannula in abdomen of patients with ascites	12	N.M.
1882	Mosso, A. and Pellacani, P.	Catheter in bladder	3	N.M.
1883	Kronecker and Meltzer‡	Tube in œsophagus	*	N.M.
1883	Schreiber, J.	Tube in œsophagus	6	N.M.
1884 and 1885	Ahlfeld¶	Balloon in rectum of children	*	N.M.
1888	Weissker, Cl.	Tube in rectum and stomach	1 (self)	N.M.
1892	Hogge, A.	Tube in fistula of pelvic cyst. Catheter in bladder. Cannula in abdomen of patients with ascites	4	N.M.
1892	Kelling, G.	Tube in stomach	6	N.M.
1895	Moritz	Balloon in stomach	5 (self and 4 others)	N.M.
1896	Kelling, G.	Tube in stomach	60	N.M.
1905	Hoermann, K.	Catheter in bladder. Tube in rectum	23	Measured
1906	Mathes, P.	*Balloon in vagina*	5	N.M.
1909	Weitz, W.	Cannula in abdomen of patients with ascites	58	N.M.
1911	Engelen	Bell jar with rubber diaphragm on abdomen	*	N.M.
1912	Kaiser, K. F. L.	Balloon in rectum	15	N.M.

TABLE I.—INTRA-ABDOMINAL PRESSURE IN MAN—Continued

Date	Author	Method	Number of subjects	Voluntary effort
1912	Idem.	Balloon in rectum	9	N.M.
1920	Peiper, A.	Balloon in rectum of children	*	N.M.
1921	Keppich, J.	Cannula in peritoneal cavity of patients about to undergo laparotomy	51	N.M.
1923 and 1924	Wildegans, H.	Cannula in peritoneal cavity of patients about to undergo laparotomy	60§	N.M.
1926	Wagoner, G. W.	Cannula in abdomen of cadavers	50	N.M.
1931	Bohnen, P.	*Balloon in vagina.* Pneumograph on chest and abdomen	90	N.M.

*Number of subjects not determined from article.
†N.M. Not Measured.
‡Quoted by Emerson (5).
¶Quoted by Engelen (7).
§Readings given in only 4 cases.

procedure resulted in significant differences in the pressures which were created.

5. The level of the reading was also affected by posture. The influence of this factor is treated in detail in the second paper of this series.

6. A full bladder or rectum obviously limited maximum effort.

RELATION OF ABDOMINAL AND VAGINAL PRESSURES

The initial inflation of the balloon was based upon the amount of air which was required to fill the vagina, as indicated by the patient's first sensation of distention. In order to secure a satisfactory figure which would represent this pressure level, a group of 29 individuals was given an average of 4.9 tests apiece. In order to eliminate the hydrostatic weight of the viscera, the vagina was inflated slowly with the patient in a 30 degree Trendelenburg posture. The limits of pressure sufficient to create the first sensation of distention varied from 1.0 centimeter to 8.7 centimeters, the average being 3.6 centimeters. Actual pain was experienced by different subjects at levels which varied from 7.5 centimeters to 10.0 centimeters. On the basis of these observations, 4.0 centimeters was selected as a suitable pressure for filling the vagina.

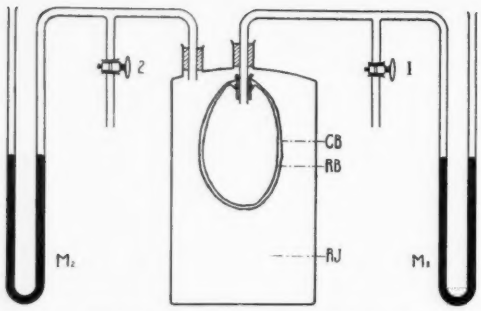

Fig. 3. Diagram of apparatus devised for testing relation between pressure created in abdomen on voluntary muscular effort, and pressure recorded in vaginal balloon, *RB*, rubber balloon; *CB*, cloth bag (simulating vagina); *RJ*, reservoir jar (simulating abdominal cavity); M_1 and M_2, mercury manometers attached to balloon and reservoir jar, respectively; *1* and *2* stopcocks.

The influence of the initial inflation pressure upon the final reading was tested on the volunteers. Three individuals, each in a different posture, were subjected to 25 tests apiece with basic inflation pressures of 2.0, 4.0, and 6.0 centimeters of mercury (Table II). The two greatest differences in this table (patients M. F. and A. W. at pressures of 4.0 and 6.0 centimeters) were selected for computation of standard errors. In each case, the actual difference between the averages fell well within the limits of 3 standard errors. Consequently, it seems that the differences among the averages resulting from tests made with these initial inflation pressures are not significant, and that such basic pressures may be disregarded in estimating intra-abdominal pressure by the vaginal balloon technique.

The relation of the pressure recorded in the vagina to true intra-abdominal pressure was also studied experimentally, by means of the apparatus shown in Figure 3. This consisted of one air system within another, each connected with its own manometer. The outer system, representing the abdomen, was a glass jar, *RJ*, approximately 25 centimeters high and 20 centimeters in diameter, with a capacity of 7,860 cubic centimeters. The inner system, representing the vagina and its balloon, consisted of a cloth bag, *CB*, and a rubber balloon, *RB*.

Basic vaginal inflation could be simulated by introducing air into the inner (balloon) system, and intra-abdominal pressures created by voluntary muscular effort by introducing air into the outer (jar) system.

TABLE II.—INFLUENCE OF INITIAL VAGINAL INFLATION PRESSURE UPON THE AVERAGE PRESSURE OF A SERIES OF VOLUNTARY STRAINING EFFORTS

Each of the 9 averages was computed from 25 tests. Note the absence of any significant effect.

Initial inflation pressure	Average pressure on effort cm. Hg.			
cm. Hg.	Patient	M.F.	A.K.	A.W.
	Posture	Semirecumbent	Recumbent	Sitting
2.0		10.9	11.7	15.0
4.0		10.6	12.2	14.8
6.0		11.7	12.1	15.7

The balloon system was inflated until manometer M_1 recorded a pressure of 1.0 centimeter and this level maintained by closing stopcock *1*. The pressure in the reservoir jar was then raised slowly (manometer M_2). Pressures in the two systems were recorded with each centimeter increment in jar pressures between 1.0 and 20.0 centimeters without altering the original inflation pressure of 1.0 centimeter in the balloon. The experiment was repeated, by means of basic balloon inflation pressures of 2.0, 3.0, 4.0, 5.0, 6.0, and 7.0 centimeters of mercury. The results are plotted in Figure 4.

The dot dash line indicates theoretical coincidence of balloon and jar pressures. The continuous line paralleling the greater part of the dot dash line represents the actual relationship between the pressures in the jar and the balloon. The parallelism and the distance between the continuous and the dot dash lines, indicate the close relationship between the actual and theoretical pressures. The distance between the 2 lines represents a difference of 0.7 centimeter between theoretical and actual pressures, i.e., the balloon pressure is 0.7 centimeter higher than the jar pressure. This increase was due to the elasticity of the balloon as indicated by the following experiment:

A rubber balloon of the type used, was connected directly to a mercury manometer and inflated until it was about 10.0 centimeters in

diameter. From the onset of the distention, the manometer registered approximately 0.7 centimeter.

It will be noted that the solid line representing actual balloon jar relationships becomes straight only after pressure in the jar overcomes the basic pressure in the balloon. The higher the balloon pressure, the higher the jar pressure had to be raised before the various curves representing basic balloon pressures of 1.0, 2.0, 3.0, 4.0, etc., centimeters joined the straight solid line. For example, with a basic balloon setting of 4.0 centimeters, all jar pressures greater than 8.5 centimeters may be read directly from manometer M_1, less the constant difference of 0.7 centimeter. In general, the balloon pressure records the jar pressure less 0.7 centimeter when the latter is a little more than twice the former. The application of this general law has previously been shown in Table II and can be tested in any subject by demonstrating that initial balloon pressures varying between 2.0 and 6.0 centimeters of mercury cause no significant change in the measured abdominal pressure.

Since in our tests of human subjects, the basic inflation pressure of the vaginal balloon was usually 4.0 centimeters, and because most of the subjects raised the mercury level above 8.5 centimeters, we conclude from the experiments on the human and the phantom that the pressures recorded in the vagina represented true intra-abdominal pressures when the latter were greater than 8.5 centimeters of mercury.

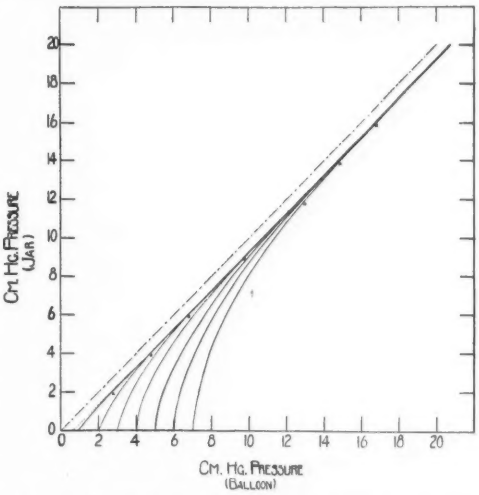

Fig. 4. Curve showing the relation between pressures (expressed in centimeters of mercury), in reservoir jar and bag shown in Figure 3. The abscissas record pressures in the cloth enclosed rubber balloon; the ordinates, pressures in reservoir jar; the dot dash line represents theoretical relation between the two pressures. The continuous line represents the actual relation and records a constant difference of 0.7 centimeter of mercury in favor of the bag system, due to elasticity of the rubber balloon. The arrows indicate the points at which the balloon pressures coincide with the jar pressures. Note: (1) that the pressures in the balloon coincide with the pressures in the reservoir jar after the initial inflation pressures of the balloon are overcome, and (2) that these points are reached when reservoir jar pressures are slightly more than twice the initial inflation pressures of the balloon.

RESULTS

A. Vaginal size. In order to gain an idea of the size of the distended vagina, anteroposterior and lateral roentgenograms were taken of 3 of the test subjects. For the anteroposterior roentgenograms, the vaginal balloon was distended with air and exposures were made using 3 levels of inflation. Figure 5 is an artist's drawing of the pelvis of R. M. (who had the smallest vagina of the subjects) when the vaginal balloon was under pressures of 2.0, 3.5, and 7.9 centimeters of mercury. It was obtained by tracing the outline of the pelvis and the balloon from three anteroposterior roentgenograms and shows the large size of the vagina in relation to the pelvis. None of the three pressures required to produce the pictured distention caused the subject any pain.

For a lateral view, it was necessary to fill the vaginal balloon with a liquid contrast medium. Figure 6 is an artist's interpretation of a lateral roentgenogram of patient A. K. The vaginal balloon and the bladder were each filled with an X-ray opaque liquid, and the rectum was distended with air. This illustration is also an accurate representation of anatomical relations and is based on a tracing from the roentgenogram. The inflated balloon is seen to extend out of the true pelvis, for its upper part rises slightly above a line drawn from the top of the symphysis to the promontory of the sacrum.

B. Measurements. A total of 1,167 tests on 5 healthy subjects was made to determine the

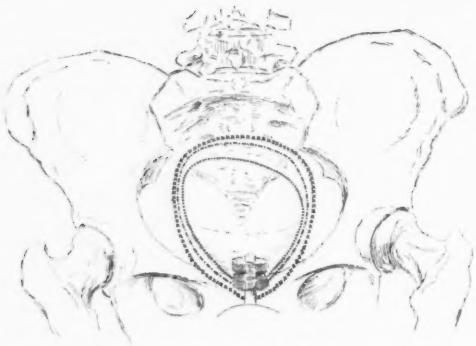

Fig. 5. Composite tracing of three anteroposterior roentgenograms of pelvis of subject R. M., when the vaginal balloon was under pressures of 2.0, 3.5, and 7.9 centimeters of mercury.

influence of posture upon intra-abdominal pressures created by muscular effort. Detailed analyses of these data are reported in the second paper of this series. The average height to which the mercury column was raised by these 5 women, irrespective of posture, and with the arms hanging free at the sides, was 13.7 centimeters of mercury—a pressure of 2.65 pounds per square inch, or 186.2 grams per square centimeter.

C. Reliability. The 1,167 readings represented 35 series of tests on 5 subjects, each tested in 7 postures. The average number of readings in a test series was 33; the lowest and highest numbers being 15 and 130, respectively. The ranges of the different test series varied greatly. The lowest was 2.4 centimeters of mercury and the highest 14.3, while the average range of the 35 test series was 6.8 centimeters of mercury.

Standard errors of 21 of the 35 averages were calculated, including the series whose ranges were smallest and largest. The standard error of the average of the test series having the smallest range was ±0.15 centimeter, while three series having approximately average ranges had averages with standard errors of ±0.35, ±0.36, and ±0.36 centimeter of mercury. Even in the test series with the greatest range and the average of which (18.1 centimeters) had the largest standard error, there was a variability of only ±0.71 centimeter. In other words, the true average of this test series probably lay between 17.39 centimeters and 18.81 centimeters of mercury.

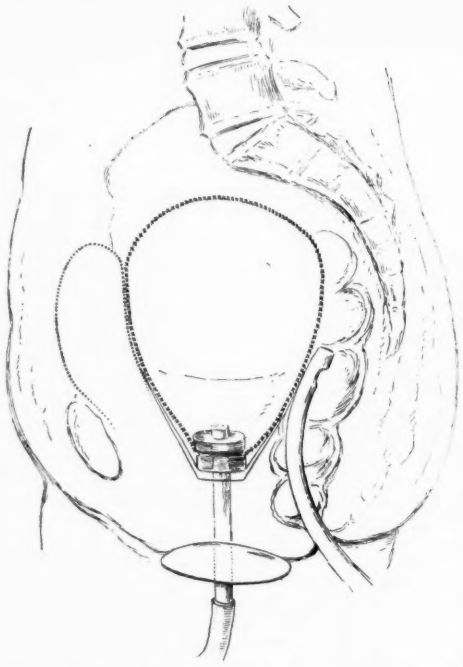

Fig. 6. Artist's interpretation of tracing of lateral roentgenogram of subject A.K. Vaginal balloon and bladder distended with X-ray opaque fluid. Rectum filled with air. Bladder outlined by dotted line, balloon by interrupted crosses. Rectum indicated by rectal tube. Note that the superior surface of the balloon rises above an imaginary line drawn from top of symphysis to promontory of sacrum.

The number of tests required to give an average within ±0.5 centimeter of mercury varies with the patient. Some of our subjects were so constant in their ability to create pressures within a few centimeters of a central point that a series of as few as 15 tests sufficed. In general, about 25 separate tests were needed in order to give a satisfactory average, while with one patient who did not co-operate well about 40 tests were necessary.

CONCLUSION

From our observations, it is concluded that the vaginal balloon technique is satisfactory for measuring intra-abdominal pressures created by voluntary muscular effort.

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CLINICAL SURGERY

FROM THE DEPARTMENT OF UROLOGY[1], NEW YORK HOSPITAL

A NEW METHOD OF REPAIRING KIDNEY WOUNDS[2]

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IT has long been known that extensive destruction to the kidney has followed nephrotomy. Most of this renal impairment can be attributed to the placing of sutures through the kidney tissue to obtain approximation of the edges of the incision and at the same time to control hæmorrhage. Sutures thus placed must necessarily compress the vessels supplying the cortical and medullary substances of the kidney and thereby produce an ischæmia with resultant necrosis and scarification.

The present paper is a preliminary report of an experimental and clinical study of the repair of kidney wounds by a new method of closure. The method was first suggested by Mr. William P. Didusch, of Baltimore and New York, in a private interview and the authors wish to give him credit for the original idea. Rabbits were the first animals used in the operations and they served the purpose well in demonstrating that the procedure was practical. The rabbit's kidney is rather small and it was deemed advisable to use larger animals in the subsequent investigations. Dogs were substituted in the second series of experiments and they have proved to be entirely satisfactory.

Before attempting the procedure upon a living human subject, it was carried out upon the kidneys of a cadaver and proved to be practical. Thus fortified, the first operation upon a human being was performed before a clinic of the Genito-Urinary Section of the New York Academy of Medicine, on February 15, 1933. Subsequently, 4 additional cases have been operated upon. The 2 earlier cases are now completely convalescent and complete protocols are included in the text of this report. The 3 latter are too recent to be included here, but as this goes to press all are in entirely satisfactory condition.

The authors have modified the method in slight details as their experience has progressed, but the original principle has remained unchanged, namely, closing the kidney by tying broad ribbon gut around the injured part of the kidney just as one would wrap a parcel with ribbon. No effort will be made herein to report the functional efficiency following this type of kidney closure. These studies are now in progress and will be reported in a subsequent paper as well as investigations of the anatomical repair of the wounds.

LITERATURE

True nephrotomy was practiced for the first time in 1880 by Morris of England, who removed a stone from a non-suppurating kidney. The patient survived and many surgeons in England, America, and Germany followed Morris' example. Among these pioneers were Le Dentu and Brodeur, in France; Morris, Bruce, Clark, Newmann, and Dickinson, in England; Kelley and Cullen, of America; Israel and Kuemmel, in Germany. Each added to the methods employed. The researches of Tuffier and the anatomical studies of Broedel, Zondek, Albarran, Papin, Delbet, and Macquot led to modifications of the classical nephrotomy incision.

The various methods of repairing nephrotomy wounds have been studied and will be briefly mentioned. Morris did not use any sutures; he merely inserted a drain into the kidney and packed around it. Tuffier made the first experimental suture of the kidney. Czerny, Porier, and Le Dentu demonstrated that renal suture was practicable. Tuffier passed a needle threaded with No. 3 catgut into the medullary substance of the kidney entirely through the sinus, placing four to six suture points in this way at a distance of 1 centimeter one from another at the start but radiating as they approached the surface.

Israel, Albarran, and Kelley all introduced methods of suture. Pappa not only sutured the kidney itself but made a veritable package of the kidney by passing a heavy thread all around it, both lengthwise and crosswise.

Hagenbach (Suter) passed a long intestinal needle through the thickest part of the parenchyma using a double catgut. Beneath the loops

[1]The Department of Urology (James Buchanan Brady Foundation) of the New York Hospital.

[2]Presented at the Meeting of the Genito-Urinary Section of the New York Academy of Medicine, February 15, 1933.

of these mattress stitches he tied down a piece of fat (corresponding to the tufts of a mattress).

Kuemmel, Jr., employed tamponage by making a tissue of resorbable catgut threads which had great hæmostatic powers. It worked well on animals but has never been used in human surgery. Rubaschow implanted the nephrotomy wound with fascia lata and fragments of muscle. Ciminata advised using fragments from the sacrolumbar mass, but neither of them made clinical use of their ideas; Joseph has employed this method on human subjects. Federoff completed hæmostasis after suture of the renal wound by fixing over the incised edges strips of perirenal fat taken from the capsule. Tschalka and Hilse and Armin (1913) all wrote on the subject of implantation of fatty tissue in closing nephrotomy wounds.

EXPERIMENTAL OBSERVATIONS

The suture material designed expressly for use in this experiment consists of flat ribbons of untwisted gut, 45 to 65 centimeters in length, 1.8 to 2.0 centimeters in width, and in thickness no more than that of fine rice paper. Packed in alcohol in the usual type of aseptic catgut tube, it remains thoroughly pliable. Though it shows a tendency to dry rapidly when exposed to air, and when dry is no longer adequately pliable, it may be readily softened again by moistening with physiological saline. In the animal experiments it has been used in bands one half the standard width, that is approximately 0.8 centimeter, but in the operations on humans the full width has been used. It has in all instances been tied in the same manner as are ordinary types of catgut; the width of the material has not interfered with adequate knots, and the tape itself has shown a tendency to twist only over a distance of about a centimeter immediately adjacent to the knot. In no instance have we noted any tendency to slip on the part of the knot. *In vitro* experiments, conducted by the manufacturers, have indicated that the tape is absorbable in 4 to 5 days.[1] Up to the present time, this has not been confirmed in our observations; the tape has been found entirely intact, though of lesser tensile strength at the end of 23 days.

In both rabbit and dog, the renal pedicle is relatively long and because of this the kidney may be readily marsupialized. The exposure, then, can be limited to a single muscle splitting incision approximately 6 centimeters in length, through which the kidney is easily deliverable. The approach in both animals is retroperitoneal; in the dog there is a reflection of visceral peritoneum on the anterior medial third of the kidney which, however, may be easily peeled off. With moderate care, therefore, it is possible in both animals to deliver the kidney completely, free of adjacent tissue and without injury to the peritoneum. In the human cases, approach has been made through the usual lumbar route.

With the kidney completely exposed, straps of kidney capsule, each about 0.5 centimeter in width, are constructed on anterior and posterior surfaces at both upper and lower poles. If the flat surfaces of the kidney be visualized as divided in equal parts by a line drawn through the hilus, the straps would lie at a point approximately in the center of each half. The straps are produced by two parallel incisions in the kidney capsule 0.4 centimeter apart and about 1.0 centimeter in length; the intervening bridge of capsule is stripped away gently by undermining it with a clamp. The direction of these incisions is parallel to that of a line drawn from hilus to pole. The actual length of the strap is decided, of course, by the width of the tape to be used. Through these straps at either pole is threaded a flat tape and the belly of the tape is looped beneath the pole; the free ends, then, meet across the lateral, convex border of the kidney proper (Fig. 1). Having made these preparations for closure, nephrostomy incision is made through the avascular line of Broedel either with the scalpel or by a strand of catgut. A clamp for grasping the calculus is thrust through into the pelvis and when the stone has been removed the closure can be completed. A small piece of freshly cut fat is introduced into the wound and the cut edges thoroughly approximated by tying the free ends of the suture tape across the line of incision. To eliminate the chance of the suture slipping off the pole of the kidney the long ends of the sutures are then tied together. In the event that this does not entirely close the wound or control the bleeding, a third tape, in the form of a figure of eight, is placed beneath either pole and crossed and tied at the midpoint of the convex border immediately over the wound (Fig. 2). In the human cases a small catheter has been left in the nephrostomy to drain the renal pelvis for 3 to 4 days. The wound is then closed in layers with plain catgut. In the animals, all the wounds have been closed without drainage; in the humans, in addition to the catheter, a Penrose sheath drain in the renal fossa has been left for 4 to 6 days.

In the animal experiments ether anæsthesia was employed. The 4 human cases have been done under spinal novocain anæsthesia; the customary dosage has been 150 milligrams.

[1]Davis and Geck, Inc., have been most generous in furnishing an unlimited supply of the material used in these experiments.

THE DATA

In the present series, the procedure has been carried out in 16 instances. The first group of operations, which was chiefly of a trial nature, was done upon 6 rabbits. Of this group, the first animal died at the conclusion of the operation. This was considered an anæsthetic death since there was no other demonstrable cause at autopsy. The second animal died at the end of 5 days, of pulmonary consolidation. The remaining animals made rapid operative recoveries and remained in excellent health; they showed no evidence of toxicity, ate well, and all but one gained slightly in weight. They were sacrificed at the end of 14, 16, 19, and 23 days, respectively. In none was there evidence at any time of suppuration in or urinary drainage from the wound.

The second group represents operations upon 6 dogs. Young animals in good health ranging from 10 to 18 pounds in weight were used. All survived in good health except one which died of general sepsis on the fourth day after operation. At the time of writing, these animals range from 13 to 39 days after operation. All animals but one have evidenced a period of toxicity beginning on the second day after operation and extending through the fourth or fifth. In this period they have been perfectly conscious but disinclined to eat or play. At the end of a week all have been as active and playful and have taken food as well as prior to operation. All the animals but one, have, except for the period of toxicity, maintained their pre-operative weight. In none has there been suppuration in or urinary drainage from the wound; in one there was a small collection of clear serum in the subcutaneous layers of the incision. None of these animals has yet been sacrificed. Nephrotomy for removal of renal calculi with closure by this method has been done very recently in four human cases. One patient has been discharged well; the second is now convalescent; and the latter two are one week after operation and doing very well.

It has seemed advisable to include in this report brief protocols of the studies to date.

Experiment 1. Closure of Nephrotomy in Rabbits

Operation 1. Right nephrotomy January 24, 1933. Moderate bleeding from kidney wound was adequately controlled by closure. Respiratory death occurred at completion of wound closure. Autopsy findings were negative. The anæsthesia was the cause of death.

Operation 2. Animal weighed 5 pounds. A left nephrotomy was done January 24, 1933. There was moderate hæmorrhage. Closure with two gut ribbons provided complete hæmostasis and approximation. Excellent recovery for 3 days. Snuffles developed on fourth day and animal died on fifth day. Autopsy showed a congested, swollen kidney evidently in the early stage of repair. There was no evident necrosis of the renal tissue. A slight amount of old blood was noted in the renal fossa. Partial digestion of gut ribbons had taken place and there was diffuse pulmonary consolidation. Cause of death: pneumonia.

Operation 3. Animal weighed 4 pounds. A left nephrotomy was done January 26, 1933. The usual closure with two gut ribbons failed to approximate the mid portion of the wound and a third ribbon was placed to close this defect. Bleeding had been moderately severe but was entirely controlled. Recovery was complete and uncomplicated. Animal was sacrificed on the nineteenth day after operation. It then weighed 3½ pounds. The kidney was normal in size, shape, and consistency. There were moderate perirenal adhesions. The gut ribbons were in place and intact. The nephrotomy wound was entirely healed; evidenced only by white scar 1 millimeter wide. No evidence of necrosis of renal tissue was noted.

Operation 4. Animal weighed 5 pounds. A left nephrotomy was done January 31, 1933. There was an usual degree of hæmorrhage. Closure was done with two gut ribbons. Hæmostasis was complete. Recovery was uncomplicated. Animal was sacrificed on twenty-third day after operation. It then weighed 5 pounds. The kidney was found in the usual position surrounded by a moderate mass of adhesions. The gut ribbons were intact and in place. The kidney was normal in size, shape, and consistency. The nephrotomy wound had healed. A small piece of necrotic fat was still present at the midpoint.

Operation 5. Animal weighed 4½ pounds. A left nephrotomy was done January 31, 1933. There was the usual amount of hæmorrhage. Closure was done by two gut ribbons, with complete hæmostasis and approximation. Uncomplicated recovery followed. Animal was sacrificed on fourteenth day after operation. Weight was then 4½ pounds. The kidney was surrounded by moderate adhesions. The gut ribbons were in place and intact. The nephrotomy had entirely healed; only visible remains were scar 1 millimeter wide and 1 centimeter in length. The kidney was normal in size, shape, and consistency.

Operation 6. Animal weighed 5 pounds. A left nephrotomy was done February 17, 1933. There was the usual degree of bleeding. Two gut ribbons were used in closure. Complete hæmostasis and approximation were obtained. Uncomplicated recovery took place. Animal was sacrificed on sixteenth postoperative day when weight was 5 pounds. The kidney was surrounded by a moderate mass of adhesions. The gut ribbons were intact and in place. The kidney was normal in size, shape, and consistency. The wound was completely healed.

Experiment 2. Closure of Nephrotomy in Dogs

Operation 1. Animal weighed 18 pounds. Intraperitoneal exploration was done, and the peritoneum was closed. Through a retroperitoneal exposure, a right nephrotomy was done February 2, 1933. There was moderate hæmorrhage. Two gut ribbons were used in the closure. Approximation and hæmostasis were complete. Hypodermoclysis was given while animal was on table. Anæsthetic recovery was complete. Animal was toxic from third to sixth day after operation, but was subsequently normal. It maintained its original weight, now 39 days after operation.

Operation 2. Animal weighed 18½ pounds. A right nephrotomy was done February 7, 1933. Brisk hæmorrhage. Closure with two gut ribbons was done. Complete approximation and hæmostasis were obtained. Hypodermoclysis was administered with animal on table. Recovery from anæsthetic and operation was rapid. There was no period of toxicity, animal was completely normal in all

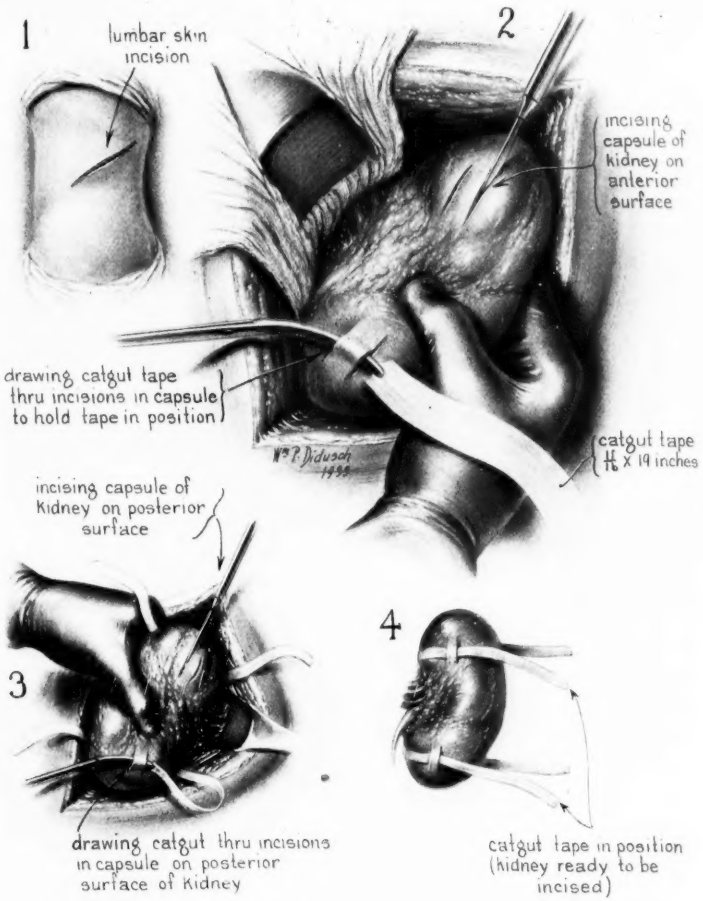
1
lumbar skin incision
2
incising capsule of kidney on anterior surface
drawing catgut tape thru incisions in capsule to hold tape in position
catgut tape ½ x 19 inches
incising capsule of kidney on posterior surface
3
4
drawing catgut thru incisions in capsule on posterior surface of kidney
catgut tape in position (kidney ready to be incised)

Fig. 1. *1*, Lumbar incision extending from the costovertebral angle obliquely down into the loin in such a manner that it is under the branches of the twelfth subcostal nerve and over the branches of the ilio-inguinal and iliohypogastric. *2*, Shows the cutting of the loops in the capsule of the kidney and drawing the ribbon gut through the loop thus made. *3*, Shows the same sort of loop made on the opposite side. *4*, Shows the ribbon gut passed through the loops on each side of both poles of the kidney ready to be tied.

respects. Moderate amount of weight was lost during first 2 weeks. Now 34 days after operation. Weight 20 pounds.

Operation 3. Animal weighed 15 pounds. Left nephrotomy was done February 9, 1933, electro-cutting instrument being used. Hæmorrhage was brisk. Closure with three gut ribbons was done, complete approximation and hæmostasis. Hypodermoclysis was administered while animal was on table. Anæsthetic recovery was satisfactory. Period of toxicity lasted from second to sixth day after operation. Thereafter animal was quite normal; maintained original weight. Now 32 days after operation.

Operation 4. Animal weighed 17½ pounds. A right nephrotomy was done February 16, 1933. There was marked hæmorrhage. The wound was closed by three gut ribbons, with complete approximation and hæmostasis. Hypodermoclysis was given while animal was on table. Satisfactory anæsthetic recovery ensued. The period of toxicity lasted from the third to the seventh day after operation; thereafter, normal. Animal maintains its original weight. It is now 25 days after operation.

Operation 5. Animal weighed 10 pounds. A right nephrotomy was done February 21, 1933. There was moderate

bleeding. The wound was closed with two gut ribbons, with complete approximation and hæmostasis. Hypodermoclysis was done while animal was on the table. Satisfactory anæsthetic recovery ensued. Animal was extremely toxic on the first day. Autopsy showed massive suppuration involving renal fossa and kidney proper, the nephrotomy incision open and draining, the gut ribbons intact and in place, no peritonitis, and superficial layers of incision free of inflammatory reaction. Cause of death was sepsis.

Operation 6. Animal weighed 13 pounds. A left nephrotomy was done February 28, 1933. There was moderate bleeding. Closure was done with two gut ribbons. Complete approximation and hæmostasis was accomplished. Hypodermoclysis was administered with the animal on the table. Recovery from operation was satisfactory. Period of toxicity lasted from second to sixth day after operation, subsequently normal. Animal maintains its original weight. It is now 13 days after operation.

Experiment 3. Closure of Nephrostomy for Renal Calculus

CASE 1. The patient is a married salesman, 40 years of age, who presented himself complaining of pain in the back of 4 months' duration. One brother was known to have suffered renal colic; otherwise the family history was irrelevant. The immediate illness dated back 1 year to the onset of painless hæmaturia, the cause for which was not determined either at cystoscopy or at suprapubic exploration. In the 4 months prior to admission the patient had had repeated attacks of left renal colic at intervals of about 1 week; similar attacks had been suffered on the right side for 2 months. On admission to the hospital, patient was free of pain and the general physical examination was quite negative except for slight residual costovertebral tenderness. The urine was turbid and of 1.012 specific gravity; albumin none, sugar none, but the sediment carried 100 leucocytes with clumps, and 25 erythrocytes per high power field. The Kline test and Wassermann reactions were both negative. Analysis of the blood chemistry showed a urea nitrogen of 17 milligrams per cent and a sugar of 92 milligrams per 100 cubic centimeters. The total excretion of phenolsulphonephthalein amounted to 38 per cent in 2½ hours. Cystoscopy demonstrated a chronically infected bladder without residual urine and grossly bloody specimens from both ureters. Pyelography demonstrated multiple renal calculi on the right and a large impacted calculus in the lower third of the left ureter.

On December 28, a left ureterotomy was done for the removal of that stone. The operative and convalescent recovery was unremarkable except for an intercurrent subcutaneous abscess of the wound at the site of drainage. During the 6 weeks' interval between first and second procedures, the blood urea nitrogen fell from 22 to 8 milligrams per cent, the excretion test with phthalein was increased from 38 to 42 per cent. Divided functional test done 2 days before the second procedure returned 12 per cent of the dye on the right and 5 per cent on the left in 1½ hours after appearance time of 4 minutes and 4 minutes respectively.

Right nephrostomy for removal of the calculi was done February 15, 1933. Anæsthesia was provided by spinal novocain. After complete evacuation of the stones, a No. 18 F. catheter was left in the kidney pelvis and the nephrostomy wound closed after the manner described. The two gut ribbons were sufficient to provide adequate approximation of the cut surfaces and likewise to establish complete hæmostasis. The kidney was returned to its position and supported by a Deming nephropexy. There was no operative shock and the patient was returned to his room in excellent condition. There was blood tinged urine draining through the catheter for 35 hours after which it became clear and the catheter was removed at the end of 48 hours. For the first 5 days, patient ran a fever which reached 102.6 degrees on two occasions; during this period the patient was clinically toxic. There was only slight discomfort referable to the kidney although there was considerable postoperative distention that responded only sluggishly to usual measures. The Penrose drain to the renal fossa was removed on the sixth day and on the next after a moderate elevation of temperature, a subcutaneous abscess was opened and the wound irrigated by the Carrel-Dakin method. Patient became afebrile on the ninth day and the Carrel tubes were removed on the twelfth postoperative day. From this point onward the wound granulated rapidly and there was no evidence of urinary leakage. Patient was up in a chair on the sixteenth day after operation. A divided renal function test for comparison with the pre-operative findings was done on the twenty-eighth day and returned 24 per cent of the dye on left and 29 per cent on right in 3 hours after intramuscular injection. There after patient was up and about ward free of complaints.

CASE 2. The patient is a married American male of 4 years, who complained of pain in the lumbar region of months' duration. Six years prior to admission patient underwent a right ureterostomy for removal of calculus and subsequently drainage of a perinephritic abscess. During the 5 months immediately prior to the present admission, the patient had experienced three attacks of low back pain associated with urinary frequency, dysuria, and pain referred to the bladder; there was no gross hæmaturia. The patient had likewise suffered a constant residual pain in the left flank during the same period. The usual physical examination was unremarkable. Laboratory analysis showed the urine to be cloudy and acid in reaction with a specific gravity of 1.028; there was no albumin or sugar, and the sediment showed many white cells in clumps. The blood sugar was 89 milligrams per 100 cubic centimeters, the blood urea nitrogen was 17 milligrams per cent and the total phenolsulphonephthalein excretion was 79 per cent in 2½ hours. Cystoscopy revealed an infected bladder without residual urine; divided functional test returned 8 per cent of the dye on the right and 6 per cent on the left over a period of 10 minutes after an appearance time of 4 minutes on both sides. Pyelography demonstrated an enlarged left kidney containing a large staghorn calculus with a smaller stone lying in the pelvis.

On March 1, a left nephrotomy over the lower pole of the kidney was done and the calculi removed. A No. 16 F. catheter was left in the renal pelvis and the nephrostomy closed with a single flat gut ribbon. This completely approximated the surfaces of the wound and controlled the hæmorrhage equally as well. The kidney was suspended by a Deming nephropexy and the wound closed in layers with plain catgut after a single Penrose drain had been left in the renal fossa. There was no operative shock and the patient returned to his room in good condition. For the first 4 hours, the patient had a marked febrile reaction amounting to 104.2 degrees F. on the first afternoon. With this febrile reaction, patient became severely distended and required gastric lavage and enemata for relief. Fluids were administered parenterally. Drainage through the nephrostomy tube was blood tinged for 2 days, but clear thereafter beginning on the second day this tube was irrigated twice daily with a solution of urinary antiseptic until its removal on the eighth day. After the fifth day the patient's vital signs remained constantly within normal limits, and he had no subjective complaints.

At the time of writing it is 21 days since operation and the patient is completely satisfactorily convalescent and he

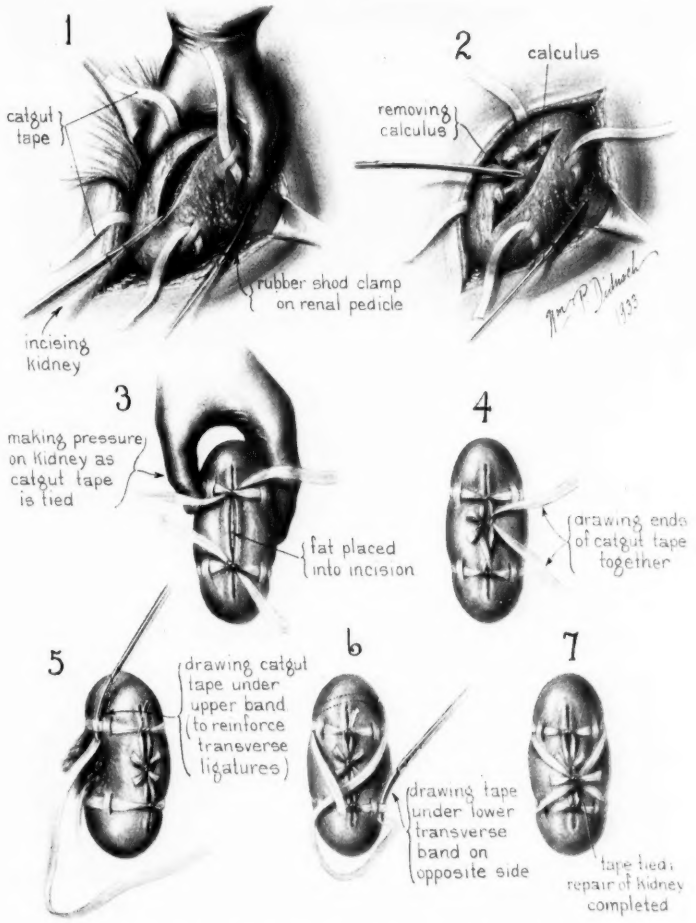

Fig. 2. *1*, Shows the incision of the kidney cortex along Broedel's line. The ribbon gut in place. *2*, Shows removal of staghorn calculus from the kidney pelvis. *3*, Shows the smooth tying across of the two ribbons, fat having been placed in the incision. *4*, Shows the tying of the loose ends of the catgut together holding it firmly in position. *5*, *6*, *7*, Show the placing and tying of catgut in such a manner that the middle of the incision is drawn tightly together.

Note.—This method has already been modified in such a manner that the knots are tied on the surface of the kidney away from the cut so that the deposit of urinary salts on the knot will not occur. It is also customary to drain the pelvis of the kidney for a day or two with a tube drain.

no subjective complaints, whatsoever. The urinary drainage ceased on the twelfth day and the wound has closed rapidly.

CASES 3 and 4. Nephrostomy for renal calculi was done 7 days before writing this article, with satisfactory immediate operative recovery.

CASE 5. Nephrostomy was done for drainage of hydronephrosis, with satisfactory operative recovery. Complete protocol will be presented later.

The foregoing facts have demonstrated that a type of closure of kidney wounds designed to eliminate the through-and-through suture is possible. It has been shown that an absorbable gut suture material which is flat and broad like a ribbon can be so placed about the kidney as adequately to approximate the cut surfaces of a

nephrotomy incision and to control all bleeding completely. In the first instance of 12 operations performed experimentally in this manner, 9 animals have lived in good health up to a period of 6 weeks. Of the 3 deaths, one is directly attributable to faulty and imperfect closure of the kidney and the 2 remaining were resultant upon factors not referable directly to the type of operative procedure. The 5 human cases have all recovered admirably well. Of the specimens observed at autopsy after intervals up to 23 days, there has been but one example of imperfect healing and that represented one in which too large a pad of hæmostatic fat had been added. In all instances the sutures have been in place and intact. These points emphasize that the procedure is not only possible but that it is permanent in its effect and that the result of such procedure is compatible with life.

Second, examination of the specimens removed at autopsy demonstrate that renal wounds closed after this manner heal in an entirely satisfactory way with a minimum of reaction as far as can be noted on gross examination. There is no gross alteration of size or consistency in the specimen.

In the third place, a description of the practical application of the technique has been offered.

Quite aware that the actual evaluation of the method depends upon the functional result in the kidney concerned, the present paper presents only such facts as have been noted to date. It is proposed to expand the experiment to include a detailed report of the effect of the procedure on the functional activity of the organ. Furthermore we are also aware that no microscopic study of the resulting scar is offered in this report. These are in progress and reports are forthcoming.

It has been noted that the suture material, though absorbable *in vitro* in 4 to 5 days, has been found unabsorbed at the end of periods up to 23 days. Since the material is non-chromicized, this fact strongly suggests that the nephrotomy wounds are tightly closed and do not leak. If there had been seepage, the plain gut would certainly have been absorbed in an interval of this length. Further, the durability of the material offers confidence that an adequate, lasting, supporting structure is afforded until such a time as the healing wound has gained its own strength.

At the outset of the experiment, there was some doubt in our minds as to whether there would be sufficient strength in the straps of kidney capsule adequately to support the gut ribbons and to keep it safely from slipping over the poles of the kidney. It is to be noted that in all the autopsy specimens the straps have been entirely intact.

The use of fat as a hæmostatic adjunct has been entirely satisfactory in our experiments. In subsequent experiments it is proposed to compare its efficiency with that of bits of muscle.

The regularity of the appearance and duration of the period of toxicity suggests that during this interval there is an inadequate elimination on the part of the kidney. The prompt and complete return to normal thereafter similarly suggests that this phenomenon is temporary and probably dependent upon local œdema within the kidney proper. The absence of infection and urinary drainage once again supports the point of view that the closure of the renal wound is complete and permanent.

CONCLUSIONS

1. Flat ribbon gut can be used successfully for the purpose of closing wounds in the kidney cortex without inserting a needle or suture through the kidney substance.

2. A wound in any part of the kidney cortex may be repaired by this method. The ribbon gut can be held in the proper location by means of small loops of the fibrous capsule.

3. It is well to tie the knot over a part of the uninjured kidney cortex in order to minimize the fibroblastic reaction present about the site of the wound.

4. Closure of a wound in the kidney cortex by this method is an acceptable surgical procedure because, first, it provides adequate approximation and, second, since it results in satisfactory anatomical repair of the kidney, and, third, because it is compatible with life and health.

5. It would seem that the principle involved could also be applied to the closure of wounds of the spleen and liver.

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BENIGN TUMORS OF THE SMALL INTESTINE

Report of Twenty-Four Cases

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RECENTLY at The Mayo Clinic, within a period of 9 months, 6 patients came to operation, at which time in each case a benign tumor of the small bowel was found to be the cause of the symptoms. This aroused our interest, for it is a universal opinion that any type of neoplasm is rarely encountered in the small intestine. In 1919, Judd noted this rarity in his report of the cases of carcinoma of the small bowel in which operation was performed at the clinic. Rankin and Mayo, in 1930, brought the report up to date. A comparative study of the benign tumors, we felt, would be instructive.

MATERIAL

Only cases in which a primary benign tumor was removed from the small intestine at operation and was proved to be neoplastic by microscopic examination of either fresh or fixed tissue are included in this report. For this reason we have omitted such cases as those of accessory pancreatic rest, carcinoid or argentaffine tumor, aberrant endometrial implant or endometriosis, retention or degenerative cyst, foreign body tumefaction, hæmatoma, secondary inflammatory polyp and polyposis, pedunculated tumor of the stomach prolapsing through the pylorus, and tumor of the ileocæcal valve, and Meckel's diverticulum. The tumors of the mesentery have been recently reported by Rankin and Major.

Thirty-five cases form the basis of this study, 11 of which have previously been reported. In 1921, Carman presented a hæmangioma of the duodenum. Camp reported a myoma of the duodenum in 1924. Balfour and Henderson, in 1929, reviewed the benign tumors of the duodenum and added 4 cases. Comfort's extensive study of submucous lipoma, in 1931, contained 1 case found at operation. Later in the same year Wellbrock described the second lipoma. In 1932, Larson reported a myxofibroma of the ileum, and Dixon and Steward, 2 leiomyomata of the jejunum. Twenty-four cases, not previously reported, are briefly summarized (Table I) and thus added to the literature. Henceforward, when we speak of "this series" and when we give statistical data in the text, we will be considering the inclusive series of 35 cases.

INCIDENCE

At the turn of the century, Heurtaux was able to collect reports from the literature of only 50 authentic cases of benign tumor of both the small and large intestine. In 1917, King found reports of 119, only 47 of which were in the small intestine. Golden, in 1928, after a complete review of the literature, found only 17 reports of cases of non-malignant tumor of the duodenum. Willis, in 1920, reviewed the records of both the Boston City Hospital and the Massachusetts General Hospital and discovered records of 19 benign tumors in 7,492 reports of necropsy. Raiford (17, 18), in a recent report, cited 37 benign tumors of the small intestine in 11,500 postmortem examinations, and was able to add 13 from the material in the department of surgical pathology at Johns Hopkins' Hospital, which included 45,000 surgical specimens.

The 35 cases in this series comprise the total number of benign tumors of the small intestine in the files of The Mayo Clinic which fulfilled the previously mentioned requirements, whereas to date, 60 patients have been found to have primary carcinomata of the small intestine.

ETIOLOGY

The etiology, of course, is unknown. Just why the small intestine is relatively free from neoplastic invasion is likewise unsettled. That there is no significant predilection for either sex is illustrated by the fact that 19 patients of our series were males, and 16, females. All were of the white race which is roughly in accord with the admissions.

Benign tumors occur among younger persons more often than do malignant tumors. The average age of patients in the present series was 38 years, whereas it was 47½ years in the series with carcinoma. The youngest patient was 14 years of age; the oldest 70. Almost 50 per cent of the patients were between 30 and 40 years of age.

No etiological factor was discovered in a study of nationality, residence, occupation, or habits.

Fig. 1. Case 3. Typical adenomatous polyp of the ileum composed of mucous glands (×115).

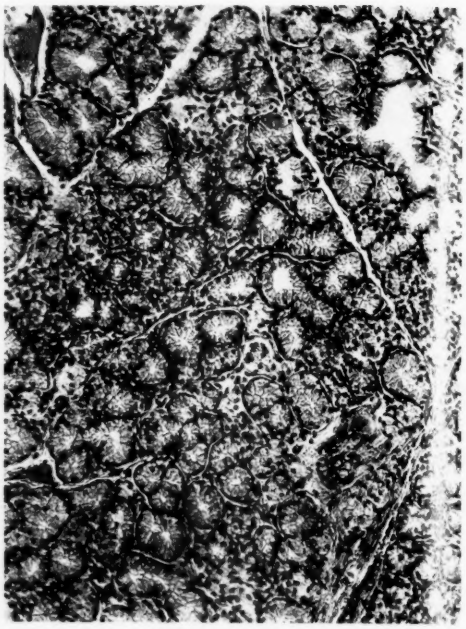

Fig. 2. Case 20. Adenoma of Brunner's glands (×115).

That 5 parents of 4 patients died from carcinoma is not considered significant.

ADENOMA

Adenomata occurred in 11 of the 35 cases. They were single in 9 cases and multiple in 2. They were sessile in 1 case and pedunculated in 10. All were intraluminal. They varied from 2 millimeters to 5 centimeters in largest diameter. Distribution was throughout the entire small bowel; 5 were found in the duodenum; 3 in the jejunum; and 3 in the ileum. Of the 11 adenomata, 9 were derived from mucous glands (Fig. 1), whereas 2 of those found in the duodenum arose from Brunner's glands (Fig. 2). One adenoma in the duodenum was the cause of severe gastro-intestinal hæmorrhage. One polyp in the jejunum and 2 in the ileum caused intussusception.

MYOMA

In the present series, myoma occurred with the same frequency as adenoma. All were single tumors, and none was pedunculated. Six of the 11 myomata were intraluminal, whereas 5 were extraluminal. The smallest was 3 millimeters in largest diameter, whereas the largest measured 6 centimeters. Six were in the duodenum; 4, in the jejunum; and 1 was stated merely as being in the small intestine. Smooth muscle cells were the outstanding element of all of them. Two of the myomata were ulcerated. In both instances they were situated in the duodenum and gave rise to severe gastro-intestinal hæmorrhage.

FIBROMA

Fibromata were noted in 6 cases of our series. In each instance they occurred as a single tumor. Two of them were pedunculated and intraluminal; the remainder were extraluminal, 2 being sessile and 2 pedunculated. In largest diameter they varied from 2 millimeters to 10 centimeters. Two were in the jejunum, 1 at the jejuno-ileac juncture, 2 in the ileum, and 1 was stated to be merely in the small intestine. Histologically, only one was a pure fibroma; the remainder of the growths had either undergone myxomatous change, had become hyalinized or calcified to such extent that it was impossible to determine whether the histogenesis was that of myoma or fibroma. In both instances in which the position of the tumor was intraluminal, the fibromata caused intussusception.

TABLE I.—SUMMARY OF TWENTY-FOUR CASES OF BENIGN TUMOR OF THE SMALL INTESTINE, NOT REPORTED BEFORE

Case	Date of operation	Age and sex	History and findings	Operation	Pathology	Follow-up information
1	7–22–12	57 M	10 year history of ulcer; obstruction last 6 months; tender right upper quadrant	Posterior gastro-enterostomy; resection of tumor of jejunum	Myoma 2 cm. diameter	Letter 9–24–28; no further trouble
2	8– 3–16	15 F	2 year history of intermittent intussusception; sausage-shaped mass	Intussusception reduced; jejunum opened; tumor removed	Pedunculated adenoma, weight 15 gm.	None; well on dismissal
3	4–15–21	34 M	8 year history of intermittent intussusception; palpable mass	60 cm. ileum resected; end-to-end anastomosis	Adenomatous polyp 5X3X2.5 cm.	Letter 5–21–25; no further trouble
4	6–30–23	33 F	11 year history of obstructive attacks; examination negative	15 cm. ileum resected; end-to-end anastomosis	3 adenomyomata; largest 2.5X2X2 cm.	Letter 7–15–28; no further trouble
5	9–10–28	37 F	7 year history of repeated severe gastro-intestinal hæmorrhages	Entire pyloric cap excised; closed as gastroduodenostomy	Ulcerating hæmangioma 4.5X4X1.5 cm.	Letter 12–17–28; no further trouble
6	10–22–28	14 M	8 months' history of intermittent intussusception; much loss of weight	30 cm. of ileum resected; end-to-end anastomosis	Polyp, 3.5X3.5X3 cm.	None; well on dismissal
7	5–21–31	54 M	18 months' history of pernicious anæmia and ulcer-like dyspepsia	Tumor excised; duodenum reconstructed	Simple multilocular cystadenoma, 2 cm. in diameter	Re-examination 7–21–31; condition excellent
8	6–6–31	32 F	2 months' history of palpable movable abdominal tumor	30 cm. of small intestine resected; end-to-end anastomosis	Pure fibroma, 10X8X8 cm.	Letter 2–17–32; no further trouble
9	6– 8–16	56 F	1 year history of pelvic malignancy	Exploration; tumor excised from small intestine	Small hyalinized fibroma	None; well on dismissal
10	12– 2–16	36 F	4 year history of chronic cholecystitis with cholelithiasis	Cholecystectomy; excision of tumor of ileum	Calcareous fibroma	None; well on dismissal
11	2–20–18	20 F	7 year history of duodenal ulcer	Posterior gastro-enterostomy; jejunum opened; tumors excised	7 polyps; largest 3 cm. in diameter	2 intussusceptions and colectomy since; well
12	6–17–20	48 M	13 year history of duodenal ulcer	Posterior gastro-enterostomy; excision tumor of jejunum	Fibromyoma, 3 mm. in diameter	Letter 2–13–29; no further trouble
13	11–16–21	70 M	40 year history of duodenal trouble with recent obstruction	Posterior gastro-enterostomy; excision tumor of jejunum	Myoma, 5X4X3 mm.	Re-examination in 1929; gastro-jejunal ulcer
14	8–17–23	52 M	2 year history of gastric ulcer and syphilis; blood and spinal fluid strongly positive	Cautery excision of ulcer; posterior gastro-enterostomy; tumor of jejunum excised	Polyp (specimen used in sections)	Died 5–24–26; cause unknown
15	4–26–24	44 F	18 months' history of carcinoma of body of uterus; uterus enlarged	Total abdominal hysterectomy; excision of tumors of small intestine	Osteochondromata; largest 2.5 cm. in diameter	None; well on dismissal
16	5– 9–24	36 M	3 year history of gastrojejunal ulcer following gastro-enterostomy	Stomach and duodenum partially resected; posterior Pólya	Adenoma, 6 mm. in diameter in duodenum	Letter 10–10–31; no further trouble
17	1–15–25	33 F	1 year history of pelvic inflammatory disease; mass in pelvis	Subtotal abdominal hysterectomy; excised tumor small intestine	Fibromyoma, 3 mm. in diameter	Re-examination 10–29–29; sacro-iliac strain (?)
18	2–17–26	31 F	Long, intermittent history of attacks of right abdominal pain	Cholecystectomy and appendectomy; tumor of duodenum excised	Myoma 4 mm. in diameter	7 subsequent admissions; anxiety, nervousness, and so forth
19	1–11–27	33 M	12 year history of bleeding duodenal ulcer	Cautery excision of ulcer; posterior gastro-enterostomy; excision of tumor of jejunum	Pedunculated fibroma 2 mm. in diameter	None; well on dismissal
20	9–14–27	41 F	6 year history of indigestion and epigastric pain	Multiple duodenal ulcers excised; pyloroplasty	Adenoma of Brunner's glands	None; well on dismissal
21	10– 7–29	38 M	6 year history of perforating and bleeding duodenal ulcer	Excision of ulcer and cap of duodenum; gastroduodenostomy	Myoma 16 mm. in diameter	Letter 1–22–30; recurrent ulcer pain
22	5–22–31	23 M	3 year history of carcinoma of ascending colon	Ileocolostomy, removal of 5 cm. ileum	Multiple polyps in ileum	Two-stage colectomy since; well 5–12–32
23	10–19–31	38 M	18 months' history of partial postoperative intestinal obstruction	Gastroduodenostomy; gastro-enterostomy taken down	Myoma 7 mm. in diameter in duodenum	Letter 6–27–32; no further trouble
24	12– 5–31	56 M	3 year history of perforating duodenal ulcer; positive Wassermann	Excised half of cap of duodenum; gastroduodenostomy	Myoma 4 mm. in diameter in wall of duodenum	None; well on dismissal

Fig. 3. Pedunculated lipoma of the small bowel (actual size). (Case reported by Wellbrock.)

LIPOMA

Only 2 lipomata were found in the present series. Reports of others tend to show that possibly they are of more common occurrence than this denotes. Morphologically, both were soft, yellow, intraluminal tumors. One was a small, sessile lipoma of the ileum, whereas the other (Fig. 3) was a pedunculated tumor 3.5 by 2.5 by 2.5 centimeters, in various diameters. Histologically, they resembled any normal fatty tissue. The pedunculated lipoma had caused intussusception.

RARER TYPES

Two hæmangiomata occurred in our series. Both were sessile and intraluminal, and both were situated in the duodenum. One was 4.5 centimeters, while the other was 5 centimeters in largest diameter. Histologically, they were composed of loose connective tissue stroma embodying blood spaces and channels of varying size. One had ulcerated and given rise to severe gastrointestinal hæmorrhage, whereas the other almost occluded the duodenum, giving rise to symptoms of obstruction.

Only 1 cyst was found which could be considered neoplastic. This was a simple, submucous, multilocular cystadenoma 2 centimeters in diameter occurring in the duodenum. Its living membrane was composed of cuboidal epithelial cells.

In 1 case there were three intramural adenomyomata of the distal part of the ileum, ranging in diameter from 1.5 to 2.5 centimeters. They were so grouped together that they caused partial obstruction. Their structure was typical.

Broders contends that fibrous tissue, wherever it exists in the body, may be the seat of origin of cartilaginous or bony tumors. If this is true the subserous osteochondromata in Case 15 (Fig. 4) were primary tumors of the small bowel, although no similar case reported in the literature has come to our attention.

SYMPTOMS

In 18 cases certain symptoms were present referable to the tumors found at operation. In the remaining 17 cases, the tumors were found incidentally in the course of intra-abdominal operations. The following observations are, therefore, limited to the former group of cases.

The chief complaint in all but 1 case was referable to the abdomen. In 8 cases it was abdominal pain; in 3 each, "stomach trouble" and hæmatemesis; in 2, palpable tumor; and in 1 case melena. The chief concern of the patient in Case 7, who was found to have both pernicious anæmia and a cystadenoma was anæmia.

In duration the symptoms varied from 2 months to 35 years, averaging 8 years, whereas for the series of carcinoma it averaged 14 months.

It was surprising to find that symptoms were essentially those of the existing complications. Obstruction in one form or another occurred in 50 per cent of the cases in which there were symptoms. In two-thirds of the cases with obstruction symptoms were caused by intussusception, whereas in the remainder they were due to encroachment of the growth on the lumen. Half of the tumors which caused intussusception were found in the jejunum and half in the ileum. The obstruction from intussusception was usually acute, complete, and intermittent, although obviously, in the chronic form it was only partial. The symptoms in all cases of intussusception were strikingly similar. Most patients had had good health until the dramatic onset of a terrific, stabbing, cramp-like pain, usually described as being in the "pit of the stomach," and not infrequently doubling the patient up. In some cases a cathartic initiated the attack. Practically all patients described a knot or lump having formed in the abdomen. Nausea, extreme vomiting, distention, gurgling, obstipation, and fever followed. Relief was finally obtained by massage of the lump, passage of gas or fæces, application of heat, or administration of opiates.

The obstruction due to encroachment on the lumen by the tumor was always incomplete, and usually both progressive and chronic. Symptoms lacked the sharply defined, intermittent characteristics of intussusception. The symptoms were of longer duration and the onset was insidious. The patients had not had good health prior to the first attack of obstruction. Constipation had long since set in, and indigestion, often of the ulcer type, especially in the presence of duodenal

tumors, had made its appearance, but, sooner or later, abdominal cramps and obstipation, with or without vomiting, developed.

Gross hæmorrhage occurred in 4 of the 18 cases; the tumor in each instance was in the duodenum. It was gastric in 1 case, intestinal in 1, and both gastric and intestinal in 2 cases. The symptoms were typical of descriptions in textbooks in each type.

It is interesting that 2 patients complained of nothing except the palpable tumor within the abdomen. Both of these tumors were large, extraluminal growths, yet they were just beginning to encroach on the lumen of the bowel.

GENERAL EXAMINATION

In 2 cases during attacks of obstruction caused by factors other than intussusception, physical examination revealed abdominal distention and rigidity, and in 1 case each, visible peristalsis and borborygmus. A sausage-shaped mass was noted in 4 of the 6 cases of intussusception. Between, or just following attacks of obstruction, residual tenderness was elicited in 5 cases, and evidence of recent loss of weight was visible in 4. The weakness and anæmia were noticeable in 2 of the 4 cases of hæmorrhage. The tumor was definitely palpable in three instances.

DIAGNOSIS

Roentgenological examination offers the only means of positive pre-operative diagnosis of benign tumors of the small intestine, and despite the fact that Waters, in 1930, was able to find in the literature only 3 instances in which roentgenological diagnosis had been made of such a condition, in the present series the roentgenologist reported benign tumors of the duodenum in 5 cases, and of the ileum in 1 case.

The extensive reviews on intussusception of Eliot and Corscaden, Watts, Willis, and others (12, 15, 16) demonstrate that the greatest single cause of intussusception of adults is a benign tumor in the small bowel. In the present series, intussusception occurred in 17 per cent of the total series and in 33 per cent of the cases in which there were symptoms, whereas in Rankin and Mayo's series of 55 cases of carcinoma of the small bowel, it occurred only twice (3.6 per cent). It is, therefore, safe to assume the presence of a benign tumor, if intussusception is found affecting an adult.

In the 10 cases of duodenal tumor, a diagnosis of duodenal ulcer was made in 4, and an associated ulcer was found in 2 of them. However, the symptoms that led to a diagnosis of ulcer were probably caused by tumor in the 2 others. In 1 case a tentative diagnosis of chronic appendicitis was made, and in another, a diagnosis of Meckel's diverticulum. It is instructive to note that the tumor in Case 8 was thought to be a pedunculated fibromyoma of the uterus.

Fig. 4. Case 15. Section from the osteochondroma of the small intestine, demonstrating both bone and cartilage in the same microscopic field (×115).

Four patients had undergone irrelevant intra-abdominal operations before admission, without relief of symptoms. These operations consisted of appendectomy in 2 cases, cholecystectomy in 1, and myomectomy in 1.

TREATMENT

Extraluminal tumors, unless of such size that their removal endangered the blood supply to the intestine, were either shelled out or dissected, usually without cutting the mucous membrane. This was done in 6 cases. Small intraluminal tumors were removed by an elliptical incision of the intestinal wall, the tumor being included in the ellipse. This applied also to pedunculated intraluminal tumors in which the attachment of the pedicle was definitely determined by palpation or by noting umbilication. If the attachment could not be determined, or if the base of the

pedicle was large, an incision was made opposite the tumor, the pedicle was clamped and ligated, or sutured, and the tumor was excised. Larger intramural tumors were removed by making a straight incision and enucleating the growths. In a majority of cases, incisions were closed transversely to increase the size of the lumen. One of these methods of excision was employed in 19 cases. In 10 cases very large tumors involving the wall of the intestine, thickened bowel that had undergone repeated invagination or bowel that had become gangrenous from intussusception, was resected with the tumor, and subsequent anastomosis was resorted to.

The entire series of 35 patients underwent operation without a death, and all were dismissed in satisfactory condition. Follow-up data were received in 14 of the 18 cases in which there were symptoms. The interval after operation varied from 3½ months to 18 years. Eight patients reported perfect results, 5 had minor irrelevant complaints, whereas one reported pain characteristic of ulcer, nausea, and one attack of inconsiderable hæmatemesis. This last patient was one of the two who had both an ulcer and a tumor of the duodenum. She was advised concerning diet for ulcer and we have received no further complaints.

SUMMARY

Thirty-five cases of true, primary, benign tumor of the small intestine are reviewed. Eleven have previously been reported, the remainder are briefly summarized, thus adding 24 cases to the literature on the subject. That these tumors are exceedingly rare is demonstrated by the fact that this series represents the total number of such tumors surgically removed at The Mayo Clinic and to date this is the largest series of its kind reported in the literature. Benign tumors were found to be twice as rare as primary carcinomata of the small bowel and afflicted younger patients. Almost 50 per cent of the patients were between 30 and 40 years of age. Adenomata and myomata were equally common in occurrence and together they comprised approximately two-thirds of the total number of tumors. Half of the remainder were fibromata. The very rare types included lipoma, hæmangioma, cystadenoma, adenomyoma, and osteochondroma. The last, as far as we know, is the first tumor of this type to be reported as having been found in the small intestine. Symptoms accompanying some of the tumors of the duodenum were like those found in cases of ulcer; in other cases, the history was of hæmorrhage. Symptoms of tumors in the jejunum and ileum were essentially those of obstruction.

Positive pre-operative diagnosis was made possible by roentgenological examination in 6 cases. Presumptive diagnosis can best be made on the basis of the following: evidence characteristic of intussusception in an adult, progressive obstruction of the small bowel, gastro-intestinal hæmorrhage, palpation of a freely movable abdominal tumor, or any combination of these.

The treatment is distinctly surgical. Not any of the 35 patients who were operated on died, although the lumen of the intestine was opened in 19 cases and resection of the bowel, with anastomosis, was employed in 10 other cases. Follow-up investigation demonstrated the obvious permanence of surgical cure.

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INJURIES OF THE MEDIAN NERVE IN FRACTURES OF THE LOWER END OF THE RADIUS[1]

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INJURIES to the median nerve in fractures of the lower end of the radius are generally regarded as of infrequent occurrence. In spite of the great frequency of fractures at this site, it is surprising how seldom involvement of the nerve is mentioned as a possible complication. Several of the leading textbooks on fractures, on orthopedic surgery, and on the peripheral nervous system, have been consulted but no specific mention of this complication has been found, except that it is referred to as an unusual injury with but little space devoted to a description of the mechanism of production. Jeanbrau states that nerve lesions, particularly those of the median, are rare in isolated cases of the fracture of Pouteau, and Wilson in his textbook mentions such involvement as a possibility.

We have no means of estimating the frequency of this complication in fractures of the lower end of the radius and we find no mention of it in the reported statistics of large series. Lewis and Miller, in 1922, surveyed, mainly from the literature, 239 cases of peripheral nerve injury associated with fractures. In this series 5 occurred in fractures of the lower third of the bones of the forearm. Of these 5, 3 involved the ulnar nerve, 1 in fracture of the lower third of the ulna and 2 in fractures of both bones. The 2 remaining involved the median nerve in fracture of the lower third of the radius.

As a contrast, with regard to ulnar nerve injuries in Colles' fracture, we have the statement of Cotton that they are not infrequent. He states that he has twice seen a total tearing of the ulnar nerve when the fracture was accompanied by luxation of the head of the ulna, but more commonly with secondary neuritis mainly of sensory type resulting from constant irritation of the nerve by the constantly slipping head. The latter, he says, in minor degree is not rare, and adds that he has seen a number of cases in which this was the major factor.

We should like to record here that it is our opinion that injury to the median nerve in fractures of the lower end of the radius is more frequent than heretofore supposed, especially a variety of injury due to treatment of such frac-

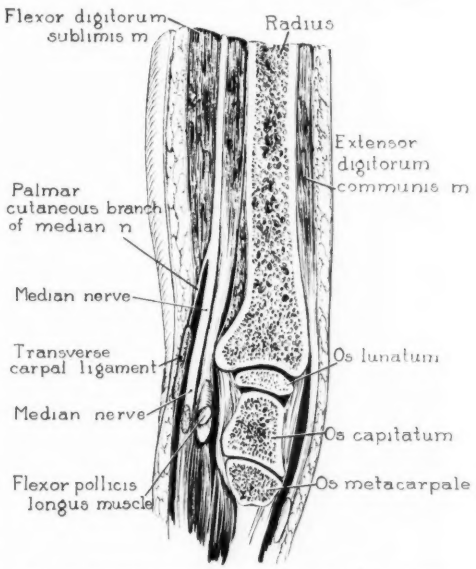

Fig. 1. Sagittal section to illustrate relation of median nerve in the region of the wrist. Note the close relationship which the transverse carpal ligament and the prominent lower anterior border of the radius bear to the median nerve.

[1]From the Divisions of Orthopædic Surgery and Applied Anatomy, University of California Medical School, San Francisco. Read before the Pacific Coast Surgical Association at meetings in San Francisco and Del Monte, February 22 to 25, 1933.

Fig. 2.

Fig. 3.

Fig. 2. Case 3. January 26, 1931. Illustrating pronounced atrophy of bones of hand and wrist.

Fig. 3. Case 3. January 26, 1931. Lateral view both wrists showing malunion with marked posterior tilting of articular surface of the radius on the right.

Fig. 4.

Fig. 5.

Fig. 4. Case 3. May 19, 1931. Position of acute palmar flexion found necessary to maintain degree of correction secured by osteotomy.

Fig. 5. Case 3. June 1, 1931. Final position after osteotomy.

Fig. 6. Effects of injection of sheath of median nerve in palmar flexion and in extension. On the right, arrest of colored solution at proximal border of transverse carpal ligament. On the left, passage of solution into palm of hand.

Fig. 7, left. Roentgenogram after injection of the sheath of the median nerve with lipiodol with the wrist held in acute palmar flexion. The solution is arrested at the level of the transverse carpal ligament.

Fig. 8. Roentgenogram after injection of the sheath of the median nerve with lipiodol with the wrist held in extension. In this position the solution flowed freely into the palm of the hand. Dispersion of the solution into the surrounding tissues was due to rupture of the sheath of the nerve during the injection.

Fig. 9. Fig. 10. Fig. 11.

Fig. 9. Case 5. July 27, 1931. Roentgenograms showing the posterior displacement of lower fragment of radius before reduction was attempted. (Courtesy of Dr. F. G. Linde.)

Fig. 10. Case 5. July 28, 1931. Failure of reduction by manipulation.

Fig. 11. Case 5. August 12, 1931. Showing postoperative reduction. Lateral view was unfortunately lost.

tures in acute palmar flexion. Our impression is that, as the fracture proper enlists the attention of both surgeon and patient and any disability which follows is ascribed to that cause, a lesion to the nerve must frequently be overlooked. Moreover, we know that in these fractures the injury to the median nerve is often incomplete and transient and may therefore never be recognized. Yet, in our experience, there have been a number of cases of sufficient severity to cause marked changes in sensation and motor power of the hand. In these more complete lesions the functional disturbance due to nerve injury has been far more important than that which resulted from the fracture. In certain instances neurolysis has been required in order to prevent a permanent crippling condition of the thumb, index, and middle fingers.

It is the object of this paper, therefore, to emphasize that in all fractures of the lower end of the radius a thorough examination should be carried out to determine whether there is involvement of the median nerve as a primary injury. Further we wish to lay special emphasis on a variety of median nerve injury which we believe to be brought about specifically by the treatment of Colles' fractures in palmar flexion, even when good reduction of the fracture has been obtained —a variety of nerve injury which we believe so far to be unrecognized. We shall also show that injuries to the median nerve, caused by palmar flexion, are due to direct pressure on the nerve between the proximal margin of the transverse carpal ligament and the distal anterior border of the radius. This fact we have determined by anatomical dissections and injections of the sheath of the median nerve. It is interesting to note that Fairbanks, in discussing a recent paper of Platt's on Colles' fracture, mentions the trophic hand which occasionally follows such fractures but expresses his ignorance of its pathogenesis. The writers or their colleagues have, during the past two years, met with more than a half dozen cases of fracture of the lower end of the radius associated with involvement of the median nerve. In the majority of these, the injury to the nerve could be directly ascribed to fixation of the fracture in palmar flexion.

LITERATURE

It would seem pertinent, at this point, to survey some of the more important articles on this subject. Though we have not been able to analyze all of the extensive literature written on these fractures yet we have reviewed available articles dating back to 1835.

The earliest recorded case we have been able to find is that of Gensoul's, 1835, in which primary injury to the nerve occurred. Paget, in his treatise on *Surgical Pathology*, 1854, quotes an interesting case of Hilton's in which the median nerve was secondarily involved consequent on a fracture of

Fig. 12. Fig. 13. Fig. 14.

Fig. 12. Case 6. May 22, 1932. Showing deformity of lower end of radius prior to reduction.

Fig. 13. Case 6. May 23, 1932. Roentgenogram showing failure of reduction by the first manipulation.

Fig. 14. Case 6. May 25, 1932. Good reduction and position of acute palmar flexion used for fixation.

the lower end of the radius which had healed with an excessive quantity of new bone. Such isolated case reports which are found mostly in French publications were collected by Blecher, in 1908. He found 9 cases and added 1 of his own. Two years later, Kirchheim wrote on this subject, as a thesis for the Friederich-Wilhelm University, Berlin, commenting upon the work of Blecher and adding 4 further cases, mostly from the literature. Since the papers of Blecher and Kirchheim we find very occasional reference to the fact that this nerve may be injured in such fractures.

Dickson illustrates a case with marked trophic disturbances and causalgia due to compression of the median and ulnar nerves in a Colles' fracture.

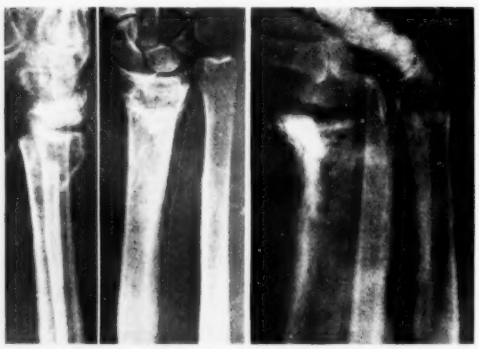

Fig. 15, left. Case 8. February 5, 1933. Position of fragment before reduction. (Courtesy of Dr. F. J. Sullivan.)

Fig. 16. Case 8. February 6, 1933. Acute palmar flexion used for immobilization after reduction.

Neurolysis was performed a year after injury with disappearance of all pain and trophic symptoms. This is the only case we have found in which both nerves were involved.

Other cases from the literature will be commented upon under the appropriate heading.

In the literature we have found no reference to injuries of the median nerve thought to be due to treatment, and it should be remembered that it is only during the last decade that the position of palmar flexion and ulnar deviation (the so-called Cotton-Loder position) has become generally popular with the profession.

CLASSIFICATION OF INJURIES TO THE MEDIAN NERVE IN FRACTURES OF THE LOWER END OF THE RADIUS

The special injuries of the median nerve which form the subject of this contribution may be divided somewhat arbitrarily into four clinical groups: (1) primary injuries, (2) secondary injuries, (3) late or delayed involvement, (4) injuries associated with treatment in palmar flexion.

The majority of these injuries are incomplete and may be classed as cases of traumatic neuritis.

Before proceeding to a consideration of these clinical types and the illustrative cases, a brief outline of the anatomy in the region of the wrist will be useful.

ANATOMY OF MEDIAN NERVE AT WRIST

The anatomical considerations of the median nerve at the wrist joint are of particular importance in explaining the special features and types

of involvement which are met with when this nerve is injured in Colles' fracture. The median nerve is accompanied in its passage from the forearm to the palm of the hand by the arteria mediana. This vessel on occasion is of substantial size and sometimes may assume such proportions as to replace the radial or ulnar artery; it anastomoses with recurrent branches of the superficial volar arch. The nerve is not directly related to the volar surface of the radius but is separated from it by the fleshy mass of the pronator quadratus muscle and by the tendon of the flexor pollicis longus muscle (Fig. 1). The pronator quadratus muscle serves to protect the nerve from fragments of this bone. Superficial to the nerve in the proximal part of its course is the flexor digitorum sublimis and the nerve is closely adherent to the deep surface of this muscle. At the wrist joint the nerve suddenly alters its course, becoming more superficial and lying on the ulnar side of the tendon of the flexor carpi radialis, behind or on the radial side of palmaris longus. Just before passing into the hand, deep to the transverse carpal ligament, it often assumes such a superficial position as to be readily palpable. So superficial may it be at this point that it may be divided by an apparently trivial cut through the skin of wrist without severance of the attendant tendons. At this point the nerve gives off its palmar cutaneous branch which supplies the proximal part of the palm of the hand with sensation. Passing deep to the transverse carpal ligament and overlapped by the lateral part of the synovial flexor sheath, it enters the palm and divides into medial and lateral divisions. Through the medium of these two divisions the nerve supplies the muscles of the thenar eminence, two or more lumbricals, and the lateral $3\frac{1}{2}$ fingers with sensation on their palmar aspect and on the more distal part of their dorsal aspect. This area of sensory supply is subject to considerable individual variation, for the median nerve anastomoses to a variable extent with the ulnar and sometimes radial nerves, connections which may explain the variability in both motor and sensory findings.

It is of the greatest importance to remember that the median nerve carries with it most of the sympathetic nerve supply of the hand, an anatomical fact associated with frequency of trophic disturbances found when this nerve is injured.

1. PRIMARY INJURIES TO THE MEDIAN NERVE IN FRACTURES OF THE LOWER END OF THE RADIUS

Primary injuries of the median nerve are those which occur at the moment of fracture and would seem to be rare. The nerve is protected by the fleshy mass of the pronator quadratus muscle and therefore its direct severance by sharp ends of bone is unlikely. The only cases we have been able to find in which the median nerve was directly involved by the bone were those reported by Gensoul and Billroth. Gensoul's patient was one with fracture of both bones of the forearm; death occurred from tetanus, and at autopsy the median nerve was found strangulated between the ends of the radius. In Billroth's case of an open comminuted fracture of the lower end of the radius, the nerve was found to be partially severed. We find no recorded case in which complete severance of the nerve has occurred.

In the majority of instances of primary injury reported, the nerve has been damaged indirectly from the pressure of a markedly displaced fragment or shaft, often combined with hyperextension of the hand as a result of the fall, so that the nerve is drawn taut over the projecting fragment (Kirchheim). Owing to the very superficial position which the nerve occupies at the wrist before passing down to the transverse carpal ligament, it may suffer a mild contusion with transient involvement of trifling character. Should the fracture be complicated by a laceration of the wrist the nerve might easily be divided.

It would seem difficult in many instances to separate primary from secondary injuries of the nerve, as such lesions may easily escape notice at the time of fracture and only be detected several weeks later, for it is the bony trauma which dominates the picture. This is particularly the case in partial injuries to the median or ulnar nerve, as there is no obvious incapacity. Contrast the readily recognized wrist drop when the radial nerve is involved.

We have met with no case which falls in this group.

2. SECONDARY INJURIES TO THE MEDIAN NERVE IN FRACTURES OF THE LOWER END OF THE RADIUS

The majority of reported cases of injury to the median nerve in fractures of the lower end of the radius are of the secondary type. We have indicated above, however, that it is difficult to assign a nerve injury to its appropriate chronological group. It is said that they may be distinguished by the clinical differences which they exhibit; primary injuries by the abruptness of onset and completeness of nerve block, secondary by the slowness of onset and by the initial incomplete nature of the lesion. But these distinctions are seldom clear and each individual case requires the

closest clinical scrutiny. It is possible that an injury initially primary may pass into the secondary class. Secondary injuries to the nerve, in the majority of circumstances, however, are due to the continued pressure of an unreduced or incompletely reduced bony fragment, or to excessive callus formation, or to both. The effect is to produce a bridge across which the median nerve is tightly strung. The combined tension and friction set up by the movements at the wrist joint inflict repeated traumata on the nerve and result in the onset of a neuritis. This is a well known cause of nerve injury elsewhere, as has been abundantly demonstrated.

Hilton's case, quoted by Paget, is the first reported case of secondary neuritis of the median nerve following fracture of the lower end of the radius and is so typical of the above mechanism as to merit re-quoting:

> A man was at Guy's Hospital who, in consequence of a fracture of the lower end of the radius, repaired by an excessive quantity of new bone, suffered compression of the median nerve. He had ulceration of the thumb and fore and middle fingers, which resisted various treatment, and was cured only by so binding the wrist that, the parts on the palmar aspect being relaxed, the pressure on the nerve was removed. So long as this was done, the ulcers became and remained well; but as soon as the man was allowed to use his hand, the pressure on the nerves was renewed, and the ulceration of the parts supplied by them returned.

On analysis of these cases, clinical phenomena occur on the average between 1 and 2 months after the fracture. Physiological nerve block ensues and it may be complete or incomplete. There is a variable wasting of the thenar musculature but motion must be carefully analyzed because the functional loss of the paralyzed muscles may be obscured by the compensatory action of other muscles. There is usually anæsthesia or paræsthesia over some part of the median nerve distribution, almost constantly over the index finger, and trophic changes in the skin are not uncommon.

We report below a fairly typical example of secondary neuritis following a fracture of the radius:

> CASE 1. Mrs. M. F., aged 47 years (Dr. F. G. Linde), sustained a fracture of the right wrist on February 21, 1932. Roentgenograms disclosed a comminuted, impacted fracture of the lower extremity of the radius and fracture of the ulnar styloid. There was no involvement of the median nerve. As this patient had had a previous heart attack and the position of the fracture was fair, reduction was not advised. The fracture was immobilized by splints which were removed in 3 weeks and physiotherapy was instituted.
>
> Four weeks after injury the patient commenced to complain of numbness in the thumb, index, and middle fingers. As symptoms were increasing in severity, she was seen by Dr. Howard Fleming who performed a neurolysis, 2 months after injury. At operation a definite thickening of the median nerve and sheath, at or just above the level of the wrist, was found. An injection into the sheath flowed down to the constricted point of the nerve. The sheath of the nerve was split and the wound closed.
>
> On the first day after operation, there was improvement in sensation. Progress was satisfactory but when last seen, 6 months after injury, there was still considerable limitation of motion in the fingers. Sensation was much improved but numbness of the fingers remained.

3. LATE OR DELAYED INVOLVEMENT OF THE MEDIAN NERVE IN FRACTURES OF THE LOWER END OF THE RADIUS

Late or delayed median nerve palsy may occur, as a rarity, in association with fracture of the lower extremity of the radius. This is illustrated in the following case. The features are essentially similar to those of delayed ulnar palsy which occurs as a remote sequela of fracture of the lateral condyle of the humerus.

> CASE 2. Male, aged 35 years, laborer, seen during routine treatment for syphilis. History given was that some 10 years previously he had sustained a fracture of his left wrist. For the past few months he had noticed numbness of the fingers which he was certain was of but recent origin. On examination there was little or no functional disability but evident deformity at the lower end of the radius. Anæsthesia or hypo-æsthesia existed over the median nerve distribution. There was obvious wasting of the thenar eminence and a slight trophic disturbance of the skin. The median nerve was not palpable at the wrist. Roentgenograms revealed an old Colles' fracture incompletely reduced with a prominence on the anterior aspect of the radius. Patient discontinued antisyphilitic treatment and, as often happens with this class of case, he was lost sight of.

Lewis and Miller report a similar case of Phemister's in which delayed involvement of the nerve occurred. The patient was a doctor who sustained a reversed Colles' fracture of the right wrist which had been imperfectly reduced. There was anterior displacement of the lower fragment. Paresis of the median nerve occurred 18 years after the accident with atrophy of the thenar eminence and moderate hyperæsthesia. The changes in the nerve were quite definitely associated with the displacement of the lower fragment.

De Rouville reports a third case of a male, aged 53 years, who, following a fracture of the radius, developed a late paralysis of the median nerve. At operation a pseudo-neuroma, three times the normal size of the nerve, was found. The end-result was not given.

4. INJURIES ASSOCIATED WITH TREATMENT OF FRACTURES OF THE LOWER END OF RADIUS IN PALMAR FLEXION

The possibility of severe damage to the median nerve by a fixed position of palmar flexion was

brought rather forcibly to the attention of one of us during the treatment of a patient whose history, clinical findings, and treatment are as follows:

CASE 3. Mrs. E. W., aged 45 years, sustained a Colles' fracture and a fracture of the styloid process of the ulna on November 13, 1930. Early treatment consisted of reduction of the fracture and immobilization for 7 weeks followed by physiotherapy to restore motion of the wrist and fingers. Two months after injury, January 26, 1931, she was referred for consultation because progress was unsatisfactory.

Examination disclosed a mal-united fracture with posterior tilting of the articular surface, obliteration of the anterior concavity of the lower radius, and a very marked restriction of motion in the wrist and fingers. Roentgenograms (Figs. 2, 3) January 26, 1931, showed faulty union with marked atrophy of the bones of the hand.

The treatment advocated was to discard splints and continue with heat and exercises. After an interval of 3 months, on April 24, 1931, very little improvement was noted, and therefore correction of the deformity by osteotomy was advised.

On May 13, 1931, an osteotomy through the site of fracture was performed. To correct the posterior tilting and maintain the articular surface of the lower end of the radius at right angles to the long axis of the shaft, a position of marked palmar flexion was necessary. In this position the hand was immobilized by anterior and posterior splints held by a single plaster-of-Paris bandage (Fig. 4).

On the day of operation the patient complained of severe pain in the entire hand. The circulation and movement of the fingers, however, was good. The day following operation she complained of numbness of the fingers, therefore the plaster was split on both sides and no constriction or pressure was noted on the anterior and posterior aspects of the wrist and hand. It was thought at this time that the numbness and lack of normal sensation was due to the position of palmar flexion, and anyone can determine for himself that maintenance of this position is accompanied by considerable numbness in the fingers. Two days after operation the patient stated that the pain and numbness of the fingers was more marked and for the first time was localized to the thumb, index, and middle fingers. It was then that suspicion arose that the cause of the disturbance was due to pressure against the median nerve. Consequently the plaster splints were freed further and the position of palmar flexion decreased by padding of the palmar aspect of the hand and wrist. At this time we were able to carry out a more complete examination which disclosed loss of sensation over the palmar surface of the thumb, index, and middle fingers and loss of power in the small muscles of the thumb. Inasmuch as there was no evidence of pressure of splints, as shown by the condition of the skin on the anterior surface of the hand and wrist, we realized that an injury to the median nerve had occurred in some other way. Our opinion was that this was directly connected with the position of marked palmar flexion. Therefore this position was gradually decreased until at the time of her discharge from the hospital on June 1, 1931, 2½ weeks after operation, anterior and posterior splints were worn with the wrist in about 15 degrees of palmar flexion. We had hoped to maintain correction of the deformity secured at operation, but we were not so fortunate as disclosed by the roentgenograms taken on June 1, 1931 (Fig. 5).

On June 18, 1931, about 5½ weeks after the operation for correction of bony deformity, the patient was seen in consultation with Dr. Howard Naffziger. Up to this time there had been no improvement in the condition of the muscles and sensation supplied by the median nerve. There was marked tenderness directly over the nerve, just above the level of the wrist. A diagnosis of a complete lesion of the median nerve was made and exploration advised.

On June 23, 1931, an operation was performed by Dr. Naffziger. An incision on the anterior aspect of the wrist showed considerable fibrosis in the region of the median nerve. More careful dissection disclosed scar tissue involving chiefly the sheath of the nerve, but also extending into its substance. An injection of salt solution distended the nerve sheath and showed that the major portion of the nerve fibers remained intact. The scar was therefore excised from the surrounding tissues and sheath of the nerve, and the wound was closed with silk sutures to the deep structures and the skin. The convalescence from this operation was uneventful and the wound was healed in 10 days. Physiotherapy was begun early with careful splinting to maintain the relaxed position of the small muscles of the thumb.

On August 12, 1931, 7 weeks after neurolysis, an improvement in the sensation of the hand was noted. On November 1, 1931, about 4 months after neurolysis, there was almost complete return in the function of the small muscles of the hand. There remained, however, marked disturbance in sensation.

On September 9, 1932, about 14 months after operation, the examination showed almost complete restoration of sensation to touch and pain with only slight atrophy of the muscles of the thenar eminence. The patient did complain, however, of some hyperæsthesia of the thumb and index finger and stated that it was difficult for her to pick up fine objects.

This patient is therefore a striking illustration of a complete lesion of the median nerve produced by marked palmar flexion following osteotomy for correction of the deformity in a mal-united Colles' fracture. This case led to dissections and finally to injections of the sheath of the median nerve to show its intimate relationship to surrounding structures. That this lesion was produced by pinching of the median nerve between the superior margin of the transverse carpal ligament and the anterior border of the lower radius was thus demonstrated in a convincing manner.

ANATOMICAL MECHANISM OF INJURY TO THE MEDIAN NERVE IN PALMAR FLEXION OF THE WRIST

There are two special features in the anatomy of this region which render the median nerve particularly vulnerable in the hyperflexed position, especially if the bone be fractured or displaced. First, owing to the sudden change in direction of the median nerve, immediately proximal to the wrist joint, from a deep to a more superficial position, it is relatively fixed and this lack of mobility prevents it from retiring from the oncoming parts in acute flexion. Second, the proximal margin of the transverse carpal ligament is sharp and resistant and lies in close proximity to the prominent anterior border of the distal end of the radius. Acute flexion, even under normal con-

ditions, pinches the nerve between these two structures as anyone who cares to do so may prove for himself by holding the wrist in acute flexion for any length of time. The tension in this region from the swelling and œdema attendant on the fracture, must render this mechanism even more effective. Injections of the sheath of the median nerve with solutions of Berlin blue and lipiodol were made with the hand held in various positions. It was found that if the hand were held in acute flexion, with some degree of ulnar deviation, the solutions were invariably arrested at a point opposite the proximal border of the transverse carpal ligament, whereas, under the same pressure, if the hand were held in moderate flexion or extension, the injections flowed easily into the palm of the hand and in some instances even along the terminal branches of the nerve. These experiments were carried out a number of times, always with a similar result (Figs. 6, 7, and 8).

FURTHER CLINICAL OBSERVATIONS ON INJURIES TO THE MEDIAN NERVE DUE TO TREATMENT

Believing at this time that similar cases to the one above cited were rare in fractures of the lower end of the radius, we were surprised to see a second patient with involvement of the median nerve evidently produced in the same manner.

CASE 4. S. W., female, had sustained a Colles' fracture on April 24, 1931. The fracture was reduced at once and immobilized in plaster for 6 weeks. Three months after the injury a diagnosis of incomplete reduction of the fragments was made, and on July 20, 1931, osteotomy was performed with immobilization of the hand in acute palmar flexion. We first saw the patient about 6 weeks after operation and she stated that following the latter there was a marked swelling of the hand with numbness of the index finger and thumb. The plaster cast was removed in 2 weeks, but no X-ray plates were taken after operation. At this time the patient complained of a marked hyperæsthesia and some loss of sensation in the thumb and index finger; also weakness of the muscles of the hand. She stated that sensation was improving gradually. Examination disclosed marked thickening of the tissues about the wrist with limitation of motion of the joint in all directions. The thumb, index, and middle fingers were of a mottled, reddish color and the sensory change was one of hyperæsthesia, particularly over the thumb and index finger. Roentgenograms showed incomplete reduction. This patient lived in Colorado and had to return there at once for further care.

We were of the opinion that this was a second case of Colles' fracture with an incomplete lesion of the median nerve which was showing definite signs of recovery.

We also learned at this time, through the courtesy of Dr. F. G. Linde, of a patient with an epiphyseal displacement of the lower end of the radius, a young lad who developed signs of incomplete disturbance of the median nerve following operative reduction of the displacement with fixation in marked palmar flexion. He supplied us with the following notes:

CASE 5. S. R., a boy 15 years of age, sustained a fracture of the distal end of the radius and styloid process of the ulna. Roentgenograms showed a separation of the radial epiphysis with marked backward displacement; also a fracture of the styloid of the ulna in good position. Four attempts at reduction by manipulation under local anæsthesia successively on July 27, 28, August 6 and 8, 1931, failed to reduce the displacement (Figs. 9 and 10).

Operation, August 10, 1931: Through a dorsal incision reduction was accomplished with ease after a loose fragment of bone had been removed. Immobilization in plaster was carried out with the wrist in a position of marked palmar flexion. Immediately following its application, it was split to insure good circulation.

The postoperative notes were as follows: August 10, circulation good; only moderate discomfort. August 11, fairly comfortable night with occasional numbness in the fingers, but this is not constant. August 12 (2 days after operation), roentgenograms show excellent position (Fig. 11). Patient is having some intermittent pain described as "deep bone pain," with motion of the first, second, and third digits. At this time these symptoms were attributed to injury of the median nerve which had resulted from trauma incurred incident to the frequent manipulations. August 15, sensation improving in the fingers. August 22, wound healed; sutures removed. The patient states that he is beginning to feel a tingling pain in the index and middle fingers. August 31, 3 months after operation, roentgenograms show good reduction with abundant callus formation. New splints applied and the patient discharged from the hospital. At this time the sensation was improving in the fingers and thumb. There never had been any definite motor paralysis. No further notes were obtainable as the patient did not return.

INJURIES TO THE MEDIAN NERVE WHEN THE FRACTURE WAS REDUCED BY MANIPULATION AND IMMOBILIZED IN PALMAR FLEXION AND ULNAR DEVIATION

Realizing that the position of immobilization was the all important factor in production of injury to the median nerve, we began to suspect that certain cases with palmar flexion after reduction by manipulation would show involvement of the nerve. In a comparatively short time we saw 3 additional cases of this type. All were incomplete lesions without any pronounced motor involvement, the principal changes being in the sensory supply of the median nerve. The chief complaint in all was pain and numbness of the thumb, index, and middle fingers. In one of these patients there was a complaint of hyperæsthesia in the fingers 6 months after the fracture had occurred.

CASE REPORTS

CASE 6. E. G., aged 44 years, was admitted to the University of California Outpatient Clinic, on May 22, 1932. Four days previously she had sustained a Colles' fracture of the left forearm. At this time the examination and roentgenograms showed posterior deformity of the distal end of the radius (Fig. 12).

On May 22, 1932, manipulation under gas anæsthesia with fixation in plaster-of-Paris splints was done. Roentgenograms showed failure of reduction (Fig. 13). Median nerve was not involved following manipulation.

On May 23, 1932, a second manipulation was carried out under gas anæsthesia and the hand was immobilized in marked palmar flexion (Fig. 14). When the patient awakened from the anæsthetic she immediately complained of severe pain and numbness of the hand. In about 20 minutes examination disclosed a decreased sensation in the fingers supplied by the median and ulnar nerves. Therefore, the cast was split along one side and no constriction of the arm was noted. By evening the hyperæsthesia cleared over the ulnar nerve distribution, but continued over that of the median nerve. On June 4, 10 days after the second manipulation, the patient still complained of numbness over the median nerve supply to the fingers. The cast was removed and a new one applied with a decreased degree of palmar flexion. On June 11, the splints were removed and motion begun. The posterior plaster was used as a splint. On June 18, the splints were removed and physiotherapy started. On October 19, about 5 months after the injury the patient still complained of numbness on the volar surface of the tip of the ring finger and thumb. Otherwise the sensation had been restored to normal.

Case 7. Mrs. H. B. T., aged 56 years, was first seen on February 18, 1932, about 6 months after she had sustained a Colles' fracture of the right wrist. The fracture was reduced and immobilized in plaster, but recurred, and a second manipulation was done with immobilization in more acute palmar flexion. Following this there was considerable pain and swelling of the fingers. She complained of numbness and severe pain in the thumb, index, and middle fingers. The immobilization was maintained for 4 weeks and was followed by diathermy and massage.

At present, 6 months after injury, she complains of limitation of motion of the wrist, weakness, and moderate hyperæsthesia of the fingers. She does not seek advice particularly about the hyperæsthesia but because of lack of rotary movements of the forearm.

The clinical examination showed that the right wrist was somewhat larger than the left. There was moderate tenderness over the lower end of the radius and over the styloid process of the ulna. Motion of the fingers and wrist was quite free, while supination of the forearm was about one-half limited. When testing for sensory involvement, we found hyperæsthesia of moderate degree over the thumb, index, and middle fingers. The patient states that this hyperæsthesia has been gradually improving.

Case 8. Mrs. E. J. (courtesy of Dr. F. J. Sullivan), was struck by an automobile on February 4, 1933, and sustained a Colles' fracture of the right radius (Fig. 15). Physical examination before reduction revealed a deep hæmatoma, about 4 centimeters in diameter, on the flexor aspect of the forearm over the distal end of the radius. The fracture was reduced and held by a plaster cast (split laterally) in extreme palmar flexion and ulnar deviation (Fig. 16). The following morning, 16 hours after reduction, the patient complained of numbness and tingling in the thumb, index, and middle fingers. Examination revealed anæsthesia of these fingers including the entire palmar surface of the middle finger. There was some limitation of motion in the thumb, notably on attempting opposition. Four days later the feeling of numbness had disappeared, the movement of the thumb had improved but there was still loss of the sensation of pain. At present, 10 days after injury, there is still hypoæsthesia over the fingers but motion of the thumb has slightly improved.

Case 9. We are indebted to Dr. H. H. Hitchcock for the following case. J. M., aged 15 years, on February 4, 1933, while playing basketball fell on the extended hand and sustained a fracture of the distal end of the left radius with complete dorsal dislocation of the distal epiphysis. He was given an anæsthetic about an hour and a half later and the dislocated epiphysis and fracture were reduced. The hand was put up in a position of acute palmar flexion to prevent slipping. X-ray films taken after manipulation showed an accurate reduction. Following fixation in this position a partial anæsthesia developed in the thumb and first two fingers. On February 15, the position of acute palmar flexion was changed to one of slightly "cock-up."

On February 20, there is still a marked diminution of sensation over the distribution of the median nerve. At the present time this is not accompanied by any pain and is apparently clearing up.

It is evident from the number of cases of median nerve injury following fractures of the lower end of the radius which we have seen, over a comparatively short space of time, that such injury is not so rare as previous work would indicate and that the matter warrants more than the passing reference which has been given it in the literature. It has been our observation that those individuals who exhibit a great deal of both initial and subsequent pain following fracture of the lower end of the radius invariably make unsatisfactory progress and show a tardy return to full function, which latter they may even fail to attain. Many of these patients exhibit trophic changes in the hand. In the past we have attributed the stiffness, notably that of the thumb and index finger, to stiffness of the joints following immobilization. While this assumption may be true in part, we feel that in many cases the delay in functional return is directly due to implication of the median nerve, either from what we have classed as a secondary neuritis or from treatment. Minor injuries to this nerve must frequently be overlooked; nevertheless injuries to this nerve though minor, may play a considerable part in the production of the incapacity which so frequently follows these fractures. When we remember how many of these patients show changes of a degree which can hardly have adequate explanation in the assumption of their being due to disuse, it is suggestive that the median nerve carries the major sympathetic nerve supply to the hand, to joints as well as to other structures.

In the section on special anatomy we have indicated the intimate relationship which the median nerve bears to the lower border of the radius and to the transverse carpal ligament. We have shown that the interval between the latter structures is strikingly diminished by palmar flexion of the wrist. Excessive swelling or the presence of a hæmatoma, by further diminishing this interval, will render nerve injury by this mechanism even more effective (Case 8). In late cases, fixa-

tion of the nerve in scar tissue subsequent to the fracture, by reducing the normal mobility of the nerve prevents its retirement from the oncoming parts.

We are of the opinion, therefore, that acute palmar flexion of the wrist should be used with great caution and in no case with marked swelling. In late cases when osteotomy is used for the correction of deformity, we advise against the use of acute palmar flexion unless the surgeon employs frequent observations to detect signs of pressure on the median nerve and is prepared to relieve such pressure by immediate change of this position.

We believe it important to emphasize here that the position of palmar flexion should be used only after complete reduction of the fracture has been secured. Yet, we have observed that this position is frequently employed in the hope that it will overcome and correct the displacement, an idea which grossly misinterprets the purpose for which this position was originally intended.

The objection may be raised that involvement of the nerve had occurred in our cases prior to the use of palmar flexion as a means of fixation. After our first experience as recorded in Case 3, we have constantly borne in mind the possibility of this complication and have made it a point for special examination before further treatment was instituted.

Considerations of the data discussed seem to emphasize the necessity of carrying out a thorough examination to determine the presence or absence of a median nerve injury with a view to prognosis and treatment. If a position of marked palmar flexion is to be used for fixation then frequent examinations should be carried out to ascertain whether there are signs of injury to this nerve. Such injury may become the major complicating factor as we have learned to our cost.

CONCLUSIONS

1. Injuries to the median nerve in fractures of the lower end of the radius may be primary, secondary, or delayed.

2. In the primary lesion the injury to the nerve is due to the initial trauma and is comparatively rare; in the secondary and delayed groups, to pressure and friction of the nerve against projecting fragments in mal-united fractures.

3. We are of the opinion that there is a fourth even more frequent cause of injury to this nerve caused by acute palmar flexion of the wrist.

4. We have found these injuries in operative reduction of old mal-united fractures and those in which the displacement is reduced by manipulation, in all associated with acute palmar flexion.

5. From anatomical dissections and injections of the sheath of the median nerve, we have shown that the injury is produced by pressure of the nerve between the transverse carpal ligament and the anterior border of the lower end of the radius.

6. The majority of the cases we believe to be incomplete and transient in character, but some are of sufficient severity to require a neurolysis. We would judge that the severity of the lesion is in direct proportion to the degree of palmar flexion.

7. In all cases of fractures of the lower end of the radius, the surgeon should determine the presence or absence of median nerve involvement before reduction is attempted. If palmar flexion is chosen as a position for fixation frequent examinations of the sensory and motor supply of the thumb and fingers should be made to avoid a possible injury to the median nerve which, in the severe cases, may lead to a degree of permanent loss of function of the hand.

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RUPTURE OF THE SYMPHYSIS PUBIS ARTICULATION DURING DELIVERY[1]

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RUPTURE of the symphysis pubis during delivery, associated with clinical symptoms, although rare, occurs more frequently than is generally believed. The lack of recognition of this lesion is a factor in its rarity. Seven cases that occurred on the Gynecological and Obstetrical Service of the Boston City Hospital and have been recently reported by the author, were subjected to various types of treatment as a means of correction. To this group are added 9 more cases of ruptured symphysis pubis occurring during delivery, the treatment of which has been carried out under a new method, which has proved more beneficial to the patient, and which has also required a shorter hospital convalescence. This method has also been utilized by the surgical services of the Boston City Hospital in the treatment of 8 cases involving fractured pelves and fractured rami, with equally efficient end-results.

In this series there were 2 primiparæ and 14 multiparæ. One primiparous rupture was due to a spontaneous delivery and the second followed forceps delivery. Of the multiparæ, 2 were in the second pregnancy, 6 in the third, 4 in the fourth, and 2 in their fifth pregnancy. One patient, who had had two previous high forceps deliveries because of high blood pressure and bagging, had a cæsarean section performed, due to the failure of the head to engage. This patient had been redelivered and re-ruptured 1 inch more in her fourth delivery. At her last examination, following a 6 months' miscarriage, she had a normal symphyseal union without separation or symptoms. Ten of these cases occurred in normal deliveries, 1 followed a breech, 2 were mid-forceps delivery, and 3 cases were due to falls on the sidewalk prior to deliveries. Twelve of these cases occurred in 6,263 deliveries from January, 1931, to April, 1933, inclusive, on the Obstetrical Service of the Boston City Hospital. The ratio of occurrence was 1 in 521 deliveries of all types.

ETIOLOGY

Many hypotheses have been advocated as an explanation for the separation of the symphysis pubis. The etiological factors fall within six groups: (1) excess of a normal physiological process; (2) trauma; (3) posture causing joint weakness or displacement; (4) subluxation of the bones due to ligamentous relaxation; (5) general lack of physical tone; (6) diseases, such as tuberculosis and hypertrophic arthritis. The increased blood and lymph supply accompanying pregnancy produces definite changes in the ligaments of the symphysis pubis as well as in those of the sacro-iliac synchondroses. Increased mobility of the pelvic girdle joints is also produced. Le Gallois in 1812, Knox in 1839, and Barlow in 1854 reported these alterations of the symphysis pubis in parturient guinea pigs, seals, and cows, respectively. Keller attributes the separation in spontaneous labor to marked intensity of uterine contractions, plus marked rapidity of labor. Duncan, in 1867, portrayed the behavior of the pelvic joints in pregnancy and labor by establishing the mobility of the pelvis, since the sacrum normally rotates within small limits on a transverse diameter. This change of the sacrum is produced in both sexes. Cantin made a study of 500 cases of relaxation of the joints during pregnancy as distinguished from rupture of the articulations during labor. He concluded that (1) relaxation of the pelvic articulation is associated with pregnancy, being most marked in the symphysis. Changes are less marked in the sacro-iliac synchondroses, but do exist. All but 2 per cent of the cases showed mobility. Sixteen per cent showed 1 millimeter relaxation, while in all others the relaxation did not exceed 3 millimeters. (2) Absolute rigidity of the joints existed in 2 per cent of the cases. (3) Relaxation occurred more frequently and to a greater degree in multiparæ than primiparæ. (4) There is no relation between the degree of relaxation and severity of symptoms which range from slight localized to referred generalized pain. (5) Pregnancy gradually increases the condition. Following delivery the condition returns to normal in a few weeks, but may persist for months or years and disable the patient to a greater or less degree. During the mechanism of labor the tilting of the sacrum or mobility of the pelvic articulations is of definite importance. The enlargement of the anteroposterior diameter at the pelvic brim by drawing the sacrum backward has been demonstrated by Walcher, for

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whom this position is named. His measurements showed that the anteroposterior diameter at the brim was greatest with the legs hanging free and narrowest with the legs flexed upon the abdomen. The diameters of the inlet and outlet can be controlled and modified within a certain range, and muscular force for expulsion can be preserved, if the displacement of the sacrum is not allowed to develop real pelvic instability.

ANATOMY

The symphysis pubis results when the anterior wall of the osseous pelvis is completed by the articulation of the bodies of the two pubic bones. It is held together by four ligaments, the anterior pubic (the strongest), the posterior pubic (the weakest), the superior and inferior pubic (arcuate). Each pubic bone is covered with a layer of hyaline cartilage. Between these hyaline layers is an interposed fibrocartilage called the lamina fibrocartilaginea interpubica, in the interior of which there is a vertical anteroposterior cleft. This cavity appears between the seventh and tenth years and is attributed to the breaking down of the interpubic lamina.

HISTOLOGICAL CHANGES

Putschar reported no principal difference in the cleft formation of the multiparæ, nulliparæ, or males in a histological study of 60 cases. Loeschcke demonstrated that the median cleft of the symphysis is often present during early life, but may be absent in males throughout life. Various zones of degeneration and vascularization appear in the hyaline cartilaginous plate which covers the bony ends, during the growth period. The disc, which is at first solid, shows, in a later period, elevations, tears, and fatty degeneration. The hyaline cartilage decreases with age and may be replaced with fibrinous cartilage. Loeschcke states that hypertrophy of the symphyseal ligaments, loosening of the symphyseal disc, the increased growth of bone in young women, and the renewed growth of cartilaginous margin in older women are the resultant severe changes produced in the symphysis by pregnancy and parturition. Termination of physiological growth of the symphysis is indefinite. Quantitative estimation of the cartilaginous growth is difficult since it is meager at 21 years and may be well pronounced at the age of 25. Cartilaginous growth is not demonstrable beyond the twenty-fifth year except for a slight proliferation of the posterior bony margins of the pubic bone. Ossification of the deeper layers of hyaline cartilage, which is not preceded by cartilaginous proliferation, is found in the pregnant, the non-pregnant, and men at this period. No enlargement of the pelvis is produced by this ossification. The degree of bony growth is calculated by the extent of forward protrusion of the ligamentous insertion which bridges the posterior surface of the symphysis. New connective tissue forms between the periosteum and bone and serves to strengthen the ligament.

PATHOLOGY

Infection, hæmorrhage, and laceration of the ligaments may be produced by this injury. The pubic ligaments are torn and the fibrocartilaginous union of the symphysis is severed. Complete separation of the joint may occur, but is rare. With infection present, abscess formation results due to lacerations extending into the vagina. Two abscesses occurred in this series, one being vaginal and the second suprapubic in character. Œdema of this area is generally due to hæmorrhage, as the result of tearing of the ligamentous fibers.

MECHANICS

The forceful descent of the fetal head through the superior strait and against the pelvic ring produces the separation of the symphysis. Sacro-iliac involvement on one or both sides and a posterior displacement of the acetabulum may often accompany this lesion. Poulett, and later Fessler, demonstrated experimentally that about 170 or more kilograms of direct pull were required to separate the symphysis. Reis et al, quoting Schatz, state that the combined contractile power of uterine and voluntary musculature is 50 kilograms or one-quarter of the force necessary as in a direct pull. This latter is probably the factor in spontaneous delivery. Sacro-iliac involvement occurred in 10 cases, being more pronounced on the left than right. Both joints were involved in 3 cases. Four cases showed no involvement.

ROENTGEN STUDIES

The evaluation of the X-ray as a diagnostic aid proved fruitless except in those cases in which there was a gross separation present. In all cases in this series diagnosis was made clinically on the symptoms, as separations, and was checked by X-ray examination as a means of noting the sacro-iliac involvement. In a separate series of normal cases following normal, forceps, and cæsarean deliveries, studied in conjunction with Dr. Paul Tivnan, although showing no increased widening of the joint following delivery, they did show motility of this joint, which persisted for 6 to 8 weeks. Fluoroscopic examination in applying this new method proved beneficial as the rupture

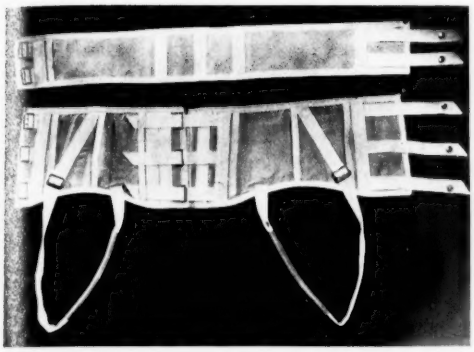

Fig. 1. Both belts are shown individually. The upper belt is the symphyseal belt.

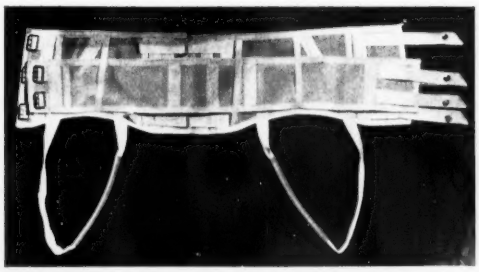

Fig. 2. Both belts in position ready for application.

could be readjusted with ease and accuracy. Flat plates were taken immediately after the application of the belt to be described and checked every 2 months in the absence of symptoms, until the belt was discarded.

CLINICAL SYMPTOMS AND DIAGNOSIS

Clinical signs of separation of the symphysis in this series occurred anywhere from 2 months before to 12 days following delivery. Pain in the symphysis region and also in the lower back, which in some cases radiated down the thigh and legs, appeared constant. Palpation of the symphysis will permit the insertion of one or two fingers between the pubic bones, if œdema and tenderness are not too marked. Vaginal examination, with the index and middle fingers in the vagina and the thumb over the symphysis, may also show the width of the separation. It is impossible for the patient to get up or lie down. Stooping is performed guardedly and is often impossible unless the knees are flexed and spasm of the hamstrings released. Marked external deviation is present if one side is affected more than the other. Lateral deviation is common. Forward bending with the knees straight is limited, as the hamstring muscles attached at the tuberosity of the ischium are made tense and by causing strain upon the sacro-iliac articulations develop muscular spasm. Lateral bending varies according to the side affected, but is more guarded on the worse side. Adduction, with the thighs flexed, develops pain and straight leg raising is limited. Spasm of the hamstrings gives rise to a peculiar gait. In taking the first step the knee can be drawn up without difficulty but as the leg straightens the spasm develops and the foot is drawn or almost jerked backward, so that in the extreme case the foot may strike the floor a little in advance of its position, when the step began.

Motility of this condition can be easily demonstrated. Forced hyperextension of the thighs, one at a time, thus moving the ilia away from the sacrum, may be sufficient. With the patient standing one hand is held over the sacrum, while the pubic bones are held between the thumb and finger of the other hand. When the patient raises one knee and then the other, the motion is often distinct. Grasping the crest of the ilia with the two hands, the thumbs resting upon the sacrum and the patient raising the legs as above, motility is often apparent. With the patient lying down straight, leg raising produces motion and pain at the symphysis.

Differential demonstration of disease of these joints can be determined by the atrophy of the muscle adjacent to or below the joint, attitude in standing or walking, by limitation in motion and by local tenderness of swelling. The character of the disease is generally determined by the appearance of the patient, and, locally, by the absence or presence of abscess or tumor formation.

TREATMENT

Removal of the tension and strain from the sacro-iliac joints, which in turn eases the symphysis condition, affords the patient considerable relief from pain. Insertion of a fracture board between the bed springs and mattress will readily relieve the usual sagging of the bed. Adhesive strapping, canvas slings enveloping the pelvis, and butterfly braces have been applied in the earlier cases and although effective, required long hospital convalescence and a constant follow-up to maintain correction of the disability. Strapping the sacro-iliac joints first and correcting this phase and then encircling the pelvis with a piece of adhesive plaster 6 inches wide afforded the best results in the methods mentioned. The difficulty encountered with adhesive was that the plaster

Fig. 3. Case to which this belt was applied. The patient exhibited all the characteristic symptoms on the tenth day after delivery. X-ray plates shows symphyseal separation with left sacro-iliac involvement.

Fig. 4. X-ray plate taken after the belt was applied and the condition corrected. The belt was applied and position obtained under the fluoroscope.

rolled, softened, and became loose after a week's body perspiration and then failed to produce an effective support. Retention of the adhesive too long also produced pustules of the skin and in one of the cases was a factor in producing a supra-pubic abscess. For the reasons mentioned, it was deemed advisable to try a belt made of sateen and involving the same principles. Under the supervision of Mr. Peter F. Rogerson, in charge of the Orthopedic shop of the Boston City Hospital, the belts are made to order and to fit the individual. Mr. Rogerson has named the belt the "Boland belt." Its efficiency has been so great in the symphysis cases that orthopedic surgeons have since used it with excellent results in other injuries to the pelvis.

Fig. 5. End-result of Figures 3 and 4 when the patient was discharged.

DESCRIPTION OF THE BELT

The belt is made of four ply sateen with the under belt 7 inches wide. The under belt portion has whale bone stays and straps that buckle in the front and back. The top belt which gives direct pressure over the symphysis is 4 inches wide and buckles in front. Felt and horse-hide pads cover the edges of the belt over the trochanter region to relieve pressure. Perineal straps which are covered with rubber tubing and which buckle in front hold the belt down in position.

APPLICATION OF THE BELT

Patients who present clinical symptoms are first roentgenographed and examined by the orthopedic service, in consultation. Measurements are taken for the belt and a temporary swathe is bound around the pelvis to give support and relieve tension. A fracture board is inserted between the bed springs and mattress. When the belt is completed the patient is removed to the fluoroscopic room in the X-ray department and the belt is applied. By tightening up on the under belt the sacro-iliacs are returned to normal position. The upper belt is then applied and the symphysis pubis is restored to normal position. During these movements the correction of the deformity can be observed accurately. A first X-ray plate is then taken for permanent record. The above maneuvers close the sacro-iliac joints,

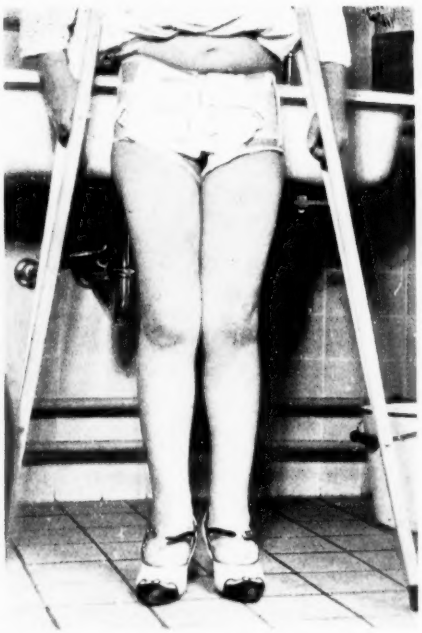

Fig. 6. Front view of the belt on the patient up and about the ward.

Fig. 7. Rear view of the belt on the same patient prior to discharge.

restore the acetabula to a normal plane, and also close the gap at the symphysis. In the first few cases in which the belt was used, severe pain was encountered when the symphysis or upper belt was tightened so as to remove the gap in this region. Further observations showed that, if the sacro-iliac separation is corrected under the fluoroscope and the upper belt is applied and tightened as much as the patient can stand, without discomfort, with the release of muscle spasm after about 48 hours, the symphysis belt can then be approximated without discomfort to the patient. The gap, if a 2 or 3 inch rupture, is then restored to normal position. The fracture board is removed as the belt supports the patient and holds the parts firmly. Crutches were used in the earlier series of cases before the belt was used when patients were allowed out of bed. Crutches are now discarded as, with the belt applied, the patient has ample support and can walk, her bowels will move, and she can bend. Patient must be free from tenderness and symptoms such as positive straight leg raising and flexion before being allowed out of bed. Many of these patients, if observed immediately following delivery, are able to be out of bed on the sixteenth day. In this last series of cases 6 were discharged in 3 weeks, 3 others in 4 weeks, as compared to an 8 to 10 weeks' convalescence under the prior treatment.

DELIVERY

Obstetricians differ as to the method of delivery when this condition is present. The writer prefers to permit nature to take its course unless there is danger to the mother or baby when delivery is then terminated by forceps. Others advocate immediate delivery by forceps or version extraction. Correction of posterior or brow positions aid the mechanism of labor.

DISCUSSION OF CASES

One patient, who sustained a separation of the symphysis, suffered a re-separation in her second delivery, but at this time restoration to normal position was more readily secured than in the first instance. Another patient, whose symptoms appeared at the seventh month, showed an elevation on the left side of the symphysis, due to a marked sacro-iliac condition also on this side. A Thomas splint with traction of 5 pounds was applied to the left leg. The belt readily relieved the sacro-iliac condition. The patient was dis-

charged after 4 weeks, free from symptoms. She returned and had a normal delivery, but separation of the symphysis was at least one-half inch more than at her first entrance. No sacro-iliac signs were present after delivery, but with the belt applied tightly following delivery, the symphysis was readily restored to normal.

Two patients had abscesses of the vulva. Morphine failed to control the labor pains in those cases in which separation occurred before delivery.

CONCLUSIONS

1. Spontaneous delivery can produce a separation of the symphysis pubis.

2. The condition occurs more frequently in the multiparous patient.

3. Pain and tenderness in the region of the pubic and sacro-iliac joints, palpable separation at the symphysis, positive straight leg raising, and peculiar gait are the most common symptoms.

4. Roentgenograms show the sacro-iliac involvement and confirm the symphysis separation.

5. Treatment consists in the use of a fracture board or Bradford frame, together with a tight swathe to relieve the acuteness of the lesion.

6. Application of the double belt, herein described, closes the sacro-iliac separation, restores the acetabula to a normal plane, and removes the gap at the symphysis pubis.

7. Re-separation of the symphysis occurred in succeeding deliveries.

8. Correction of the symphyseal and sacro-iliac separation are essential for functional results.

9. The double belt permits a shorter hospital convalescence and restores earlier function than other methods.

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RE-ESTABLISHMENT OF NORMAL LEVERAGE OF THE PATELLA IN KNEE FLEXION DEFORMITY IN SPASTIC PARALYSIS

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FLEXION deformity of the knee in spastic paralysis presents a problem not to be solved by any one surgical measure. Preservation of calf action in counteracting flexion of the knee is well recognized. In spastic paralysis, this action corresponds to the stabilization, by calf action, of flaccid paralysis involving the quadriceps. Manipulative correction of flexion deformity of the knee in spastic conditions is seldom successful. Division and lengthening of the hamstring muscles or division of the motor nerve supply of these groups is frequently sufficient to permit standing with the knees in full extension. At times, transplantation of the biceps to the anterior aspect of the thigh to act as an extensor, aids in maintaining extension.

In a limited group of spastic cases none of these procedures is successful and weight bearing with the knees completely extended is impossible. Such patients stand and walk with the knee in partial flexion (Fig. 9). Active extension to about 160 degrees is possible and, in some, complete passive extension is possible, especially if the hamstrings have been lengthened. In these cases the position of the patella should be determined.

The normal patella is in a position anterior and distal to the epiphyseal line at the lower end of the femur (Fig. 8A). On flexion, the patella fits into the intercondylar groove and diminishes in prominence as flexion becomes more pronounced (Fig. 10B). If the spastic patients who stand with knees partially flexed are examined to ascertain the position of the patellæ, some will be found to depart widely from the normal. The patella will be found opposite the lower shaft of the femur and proximal to the articulating surfaces of the condyles. On flexion, these patellæ become prominent, riding high on the condyles of the femur, giving the knee an angular appearance (Figs. 5, 6A, 7A, 10A, 10C). The patellæ do not sink into the intercondylar notch. The patellar tendon appears unusually long and is prominent anteriorly. Active extension is limited even when the posterior muscles have been lengthened. With the patella in this position, quadriceps action is lost throughout the last few degrees of extension because of the loss of leverage normally transmitted through it.

The elongation of the patellar tendon and the secondary high position of the patella may be considered adaptive changes resulting from prolonged tension during the period of rapid growth. In complete extension of the normal knee while weight bearing, the quadriceps muscle is relaxed. Contraction begins, however, within the first few degrees of flexion and continues as long as weight is carried through the knee joint. In some spastics, the knee is never completely extended; consequently, there is no period of muscular relaxation while weight bearing. This continued tension added to the spastic tonicity of the muscle may readily result in elongation of the patellar tendon.

After many unsuccessful attempts to correct this type of flexion deformity by stretching of the knee, releasing of the posterior musculature, and by transplanting the hamstring muscles, the following operation to restore the normal leverage of the patella was developed.

TECHNIQUE OF OPERATION

A vertical lateral patellar incision angulating medially across the center of the patellar ligament and extending distally along the anteromedial aspect of the tibia, is made through the skin and subcutaneous tissues. Parallel incisions along the medial and lateral margins of the patellar tendon expose its entire length. The tendon separates from the anterior fat pad very readily and its insertion into the tubercle of the tibia is exposed. Then a block of bone at the insertion of the tendon is removed. A second block of tibial cortex, distal to the first and separated from it by an in-

Fig. 1. Photograph of knee with elongation of patellar tendon. Note angular contour and high patella.

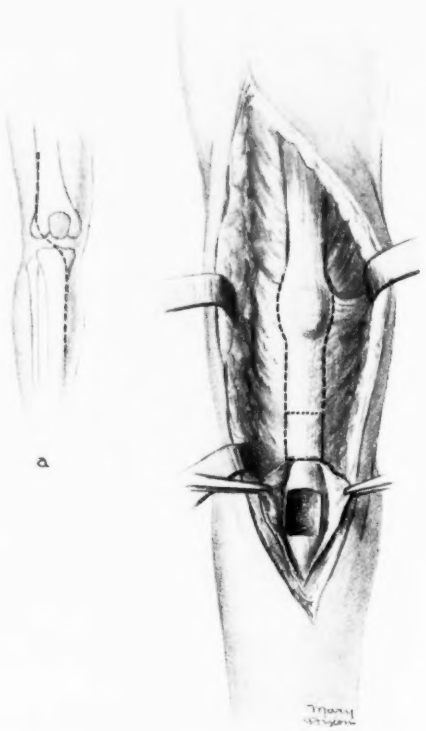


Fig. 2.

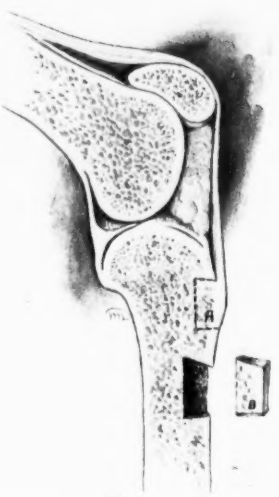


Fig. 3.

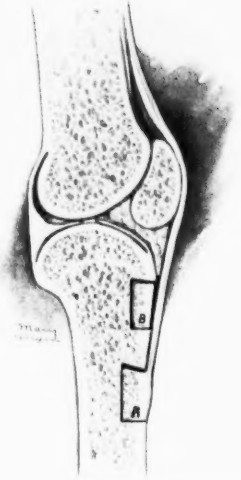


Fig. 4.

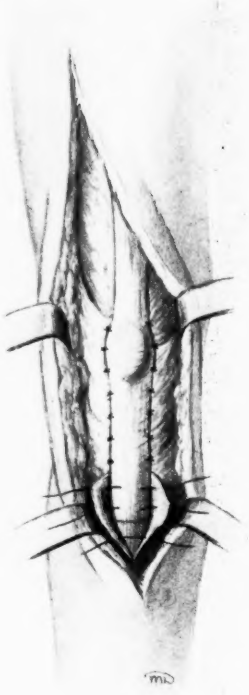


Fig. 5.

Fig. 2. *a*, Incision. Patellar tendon is freed by incision through fascia and periosteum as indicated by heavy dotted lines.

Fig. 3. Sagittal section through knee before advancement of insertion of patellar tendon. *A* is then transposed to distal recess in tibial surface.

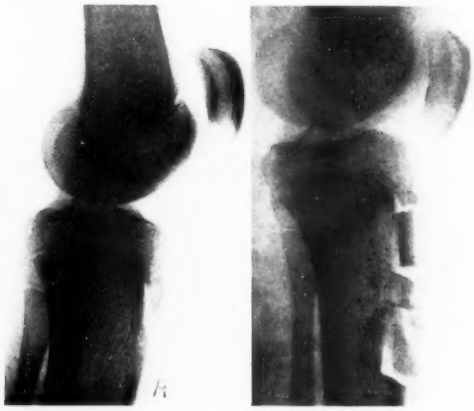
Fig. 4. Sagittal section of knee after advancement of patellar tendon. Bone fragment *B* filling defect left by removing *A*.

Fig. 5. Closure. Periosteum mattressed over transplanted insertion of patellar tendon. The aponeuroses are then sutured to the tendon and patella.

A. B.

Fig. 6. A, Case of severe spastic paraplegia showing elongated patellar tendon with high position of patella. B, Lateral roentgenogram of knee following advancement operation.

tact ridge of cortex, is removed (Figs. 2 and 3). The patella is mobilized by division of the aponeuroses of the vastus medialis and vastus lateralis, and the tibial tubercle is countersunk into the second recess in the tibia, thus moving the point of the insertion of the patellar tendon distally about one and one-quarter inches. The distal block may be replaced in the cavity remaining after the removal of the tubercle. The tendon and insertion then are fixed in their new position (Figs. 4, 5, 6, and 7.)



A. B.

Fig. 7. Before and after advancement of patellar tendon.

This advance of the patellar tendon may be accomplished without opening the knee joint. The preliminary division or lengthening of short hamstring muscles is essential.

The leg is kept fully extended in a plaster splint for 8 weeks. Active and passive movements are then begun.

A. B. C.

Fig. 8. A, Lateral roentgenogram of normal knee flexed 90 degrees. Note position of patella in relation to condyles. B, Lateral roentgenogram of left knee before advancement operation was performed. C, Lateral roentgenogram showing appearance of left knee after advancement operation was performed.

Fig. 9. Patient E. P. Showing adduction of thighs, flexed position of knees, and high patellæ.

By bringing the patella down to a normal position, the leverage of the quadriceps femoris is reestablished and full extension of the knee made possible.

CASE REPORT

E. P., female, aged 9 years, admitted to Children's Memorial Hospital, February 23, 1932. The patient complained chiefly of inability to use arms or legs; inability to talk; total inability to walk. Patient was a full term baby with shoulder presentation. This was converted to a cephalic presentation and delivery was accomplished by instruments. At time of delivery, the head was badly distorted and there was some laceration of the scalp. Deep cyanosis was present and resuscitation very difficult. Twenty-four hours after birth there were two severe convulsions but none since that time. The child did not cry until about 6 months of age. She has been unable to sit up on a table or in bed, but can sit up in a chair. She has never walked. The head was held in a flexed position until she was about 5 years of age, when she began to extend it actively. She made attempts to talk when 6 years old, but could not say words which could be understood. At about 3 years of age, she showed the first signs of being interested in toys. Besides the general spastic condition of all muscles, she has had no serious illness, with the exception of bronchopneumonia during infancy.

The father and mother are alive and well. There is no history of one other child who died at birth following a forceps delivery.

Admission laboratory findings: red blood cells, 4,300,000; hæmoglobin, 76 per cent; white blood cells, 9,300; polymorphonuclears, 44 per cent; lymphocytes, 56 per cent; urinalysis, negative; von Pirquet, negative; Wassermann negative.

Examination revealed a poorly developed, poorly nourished white girl of about 9 years of age, not acutely ill. The patient was unable to sit up or talk. There was a very evident spasticity of marked degree involving all extremities, facial and trunk muscles. The eyes were normal except for some spasticity of motion and some dilation of the left pupil. Both reacted normally to light. The heart, lungs, and abdomen were essentially normal. The upper extremities showed a very marked spasticity with the arms held partially flexed at the elbow, and the forearms were somewhat pronated. Irregular jerky movements were present in both arms, the patient making desperate attempts to cooperate in producing a purposeful movement. The head was held partially flexed and, on attempting to look up, had an irregular rolling motion to both the right and left sides. Patient was unable to sit except when bolstered up in an arm chair. She was totally unable to stand. When assisted to a standing position, both legs crossed in an extreme scissors position. The knees were flexed 25 to 30 degrees, and the feet were in marked equinus. All reflexes were hyperactive. There was a bilateral Babinski.

A. B. C.

Fig. 10. A, Lateral view of right knee in flexion. Note angular contour of knee caused by high position of patella on condyle. B, Lateral view of normal knee showing depression of patella into intercapsular groove, on flexion. C, Position of patella when active extension is assisted. The patellar tendon is elongated.

Operation was done on March 12, 1931. The hamstring muscle groups of both knees were divided and extension casts were applied. This resulted in complete correction of the flexion deformity. On March 25, 1931, lengthening of both Achilles tendons was done. The patient was given general exercises directed at the development of better co-ordination and was given speech training. Very satisfactory correction of flexion deformity of the knees and equinus position of the feet was obtained. The patient was able to walk between parallel bars but was unable to stand without assistance. Under weight bearing, the knees flexed to a position of about 25 degrees. The tendency to a scissors gait persisted. The patient was discharged home and at a later date was admitted to the Spalding School into a class for spastic children.

In August, 1932, the patient was re-admitted for further surgery. During this time, there had been a very definite improvement in speech and general co-ordination of the upper as well as the lower extremities. The scissors gait was very pronounced. This was corrected on August 16, 1932, by a bilateral suprapubic extraperitoneal resection of the obturator nerves. Immediate and complete relaxation of the abductor spasm was secured. Sufficient active adduction persisted to enable the patient to bring knees together voluntarily. Although passively both knees could be extended completely, when weight bearing or in attempts at voluntary extension, 25 to 30 degrees flexion persisted. At this time, it was noted that both patellæ rode very high on the condyles, giving both knees an angular appearance. It was obvious that both patellar tendons were markedly elongated and that all the leverage through the patella was lost during the last 25 degrees of extension.

On September 15, 1932, an advancement operation was done, the insertion of the patellar tendon on the left being advanced one and one-quarter inches. Casts were applied, holding complete extension for 8 weeks. This was followed by active and passive movement to restore motion at the knee joint. Under weight bearing and active contracture of the quadriceps muscle, complete extension was possible. On January 17, 1933, a similar operation was repeated, advancing the insertion of the patellar tendon on the right, one and one-quarter inches. This was followed by retention in plaster in a fully extended position.

CONCLUSION

Elongation of the patellar tendon with loss of patellar leverage is encountered in spastic paralysis.

The re-establishment of the normal leverage of the patella permits complete voluntary extension of the knee joint.

Waltham, in 1893, employed this principle in the treatment of lateral dislocation of the patella.

TREATMENT OF FRACTURES OF THE HEAD AND NECK OF THE RADIUS AND SLIPPED RADIAL EPIPHYSIS IN CHILDREN[1]

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WE have recorded 64 references from the literature, 34 of which have been reviewed to form a basis for our conclusions. These indicate that the subject of this paper is no longer to be regarded as a rare traumatic affection.

Our purpose, therefore, is to summarize the present status of treatment of fractures of the head and neck of the radius. Special attention is directed toward the question of immediate operation for correction of deformity in cases presenting displacement of the radial epiphysis and malalignment in fractures of the radial neck. Three cases are reported to illustrate these two types of fractures.

The mechanism of injury is usually characterized by a fully extended and pronated forearm which receives the body weight in protecting the patient from a fall. A blow received over the radial head and neck may produce such an injury.

The following *classification* forms the basis for the subsequent consideration of the various forms of treatment.

A. Fractures within the radiohumeral joint—1. Fractures of the radial head. 2. Displacement of the radial epiphysis. 3. Linear fractures from the radial head into the radial neck.

B. Fractures outside of the radio-humeral joint—1. Fractures primarily affecting the radial neck between the level of the synovial membrane attachment and the insertion of the biceps tendon.

C. It is understood that varying degrees of comminution, impaction and malalignment may prevail in either the A or B group. Moreover, group B is frequently complicated by conditions prevailing in group A. Such fractures may be complicated by varying degrees of injury to the humerus, particularly fracture of the external condyle. The presence or absence of injury to the radius should be sought in all cases of elbow dislocation.

The *clinical picture.* The forearm is usually supported by the opposite hand, in a position of approximately 90 degrees flexion at the elbow. Muscle spasm holds it in a position midway between pronation and supination.

There is the usual evidence of periarticular tissue reaction to trauma. The maximum swelling with varying degrees of ecchymosis is usually centered over the radial head. The external evidence of injury, in the absence of deformity, is usually not great enough to explain the disability present. Gentle passive extension and flexion of the elbow may be 50 per cent of normal with relatively little evidence of pain. Although this is a variable finding, great pain accompanies any passive effort to change the degree of pronation or supination from the fixed position. Radial deviation of the forearm causes greater pain more quickly than when a similar passive effort is made to produce ulnar deviation. Palpation gives evidence of tension due to underlying hæmorrhage. The application of the index finger may define a point of maximum tenderness over the radial head and neck. All three of the patients reported herewith complained of referred pain to the radial side of the wrist when pressure was applied over the radial head and neck by the index finger. This clinical observation has not been recorded in any of the references reviewed by the authors. Crepitus is frequently absent regardless of methods employed to elicit it; moreover, this physical sign is not essential to the clinical diagnosis. As a general rule, altered alignment of the fragments may be neither seen nor felt.

Roentgenographic examination. The radial epiphysis does not ossify until the fifth to the seventh year and unites at the age of 18 to 20 years. Anteroposterior and lateromedial views should be made with the normal forearm held in the same degree of flexion and supination as the affected side. Rarely, chip and linear fractures of the radial head may not be revealed. Gross evidence of intra-articular and extra-articular injury of the radius prevails in roentgenograms of all other fractures included in both A and B groups.

Historical background since the time of Hippocrates. Without the intention of being all-inclusive, the following opinions of 34 authors have been expressed by quotations from respective papers for the obvious advantage of the reader in judging our conclusions.

[1]Read before the Robert Jones Orthopædic Society, November 5, 1932.

We were unsuccessful in finding reference to these fractures in the works of Hippocrates (460–370 B.C.). Paul of Aegina (625–690 A.D.) may have known of this injury to the radius: "The ulna and radius are sometimes both fractured together, and sometimes one of them only, either in the middle or at one end, as at the elbow or wrist." His brief subsequent discussion does not include further reference to the radial head and neck.

Petit (1726) described a method of eliciting crepitus:

It is essential to hold the upper Part of the Fore-Arm with one Hand, whilst with the other one makes the Hand of the Patient alternatively perform the Motion of Supination and Pronation; and if at that Time the Radius is found to resist the Hand that holds the upper Part, and makes an Effort against it to move in Pronation or Supination, one may be assured that there is no Fracture. On the contrary if the Bone is broken, it will not resist, and a Crepitation will be heard, because the under Part of the Radius which is moving, will rub against the upper Part, which is kept, as it were, by the Hand that held the upper Part of the Fore-Arm.

This and the remainder of his discourse fails to indicate that Petit recognized fractures of the head and neck of the radius.

Nearly one hundred years later (1811) found Desault writing as follows.

In whatever way the fracture may be produced, it occurs in the middle or at the extremities of the bone; very rare near its articulation with the os humeri, it is more common in the middle; but more frequent still at its lower end.—If the fracture exists at the upper end, the thick muscular covering which there surrounds the radius, renders the diagnosis more difficult.

He then referred to Petit without giving further information on this subject.

In 1830, S. D. Gross, of Philadelphia, cited Petit; he almost repeated but did not mention Desault, and contributed nothing original.

In fractures of the superior extremity of the radius, the symptoms are generally more faintly developed, on account of the great number of muscles in which the bone is imbedded, and the diagnosis is consequently more difficult.

James Syme (1832) made no mention of the possibility of injury following direct or indirect trauma to the radial head and neck.

The first definitely recorded observation of fracture of the radial head followed an accidental postmortem finding by Berard, in 1834.

Mais ce signe n'est point aussi infaillible que le pense M. Dupuytren. J'ai vu cette année (1834) à l'hôpital Saint-Antoine, le bras d'un individu qui s'était tué en se jetant d'un second étage sur le pavé; le coude gauche était le siége d'une déformation en tout semblable à celle que l'on observe dans la luxation. La réduction fut tentée et opérée sans trop de difficultés, quoiqu'il y eût de la rigidité cadavérique; une pression médiocre, exercée sur l'avant-bras et le bras en sens contraire, suffit pour opérer un nouveau déplacement, qui s'accompagna d'une légère crépitation. Ces manoeuvres de réduction et de luxation furent accomplies plusieurs fois de suite avec le même résultat. Les caractères assignés par M. Dupuytren à la fracture transversale de l'humérus étaient donc ici on ne peut plus évidens. Or, voici ce que la dissection a montré: 1° luxation de l'avant-bras en arrière; 2° fracture d'une partie de l'apophyse coronoïde du cubitus; 3° fracture d'une portion du radius, qui divisait la cavité articulaire de cet os de dedans en dehors, et aboutissait à un demi-pouce de son extrémité supérieure sur la face antérieure, d'où résultait un fragment triangulaire qui, par son déplacement facile, enlevait à la cavité articulaire du radius un tiers à peu près de sa surface.

This was not a simple fracture of the radial head, a fact not emphasized by previous references to Berard's report.

Ten years later (1844) Sir Astley Cooper said that fractures of the neck of the radius were "mentioned by surgeons as being of frequent occurrence, but there must be some mistake in the statement, for it is an accident which I have never seen; and if instances ever present themselves (which I do not deny), they must be very rare."

Helferich (1897) gave the first unquestionable evidence of recognition and treatment of fractures of the head and neck of the radius together with epiphyseal displacement.

Fracture of the head.—This is wholly intra-articular; it may be complete or incomplete (fissure or bending). In the latter case the diagnosis is naturally difficult and uncertain. Cases of complete fracture are to be recognized when the head of the radius is abnormally movable with crepitus, but it may be noted that in such cases the movement of the head in pronation and supination seems to be unaffected. Pain is naturally localized to the region of the radial head. This fracture may be due sometimes to direct, more frequently to indirect, violence, and the elbow may be either extended or flexed at the time. There are naturally present the signs of injury to the joint, and not infrequently this fracture is overlooked, and considered to be only a contusion or a distortion of the elbow. Occasionally the radial nerve is damaged at the same time. Since one can make no direct pressure on the small upper fragment, union will probably take place, in spite of all precautions, with considerable deformity and limitation of movement, which may later justify operation and resection of the radial head. Fractures of the neck of the radius and traumatic separation of the upper epiphysis are both extremely rare, as also are fractures of the radial shaft alone.

He gives two illustrations of fracture of the radial head united by bone. The specimen was resected from a woman aged 28 years, who had fallen on the outstretched hand. The joint was stiff at an obtuse angle and pronation was limited.

In 1900, Mouchet reported 11 cases of fracture of the radial neck in children.

The prognosis is good in these cases if the fracture has been properly treated at the beginning. As soon as a diagnosis is made it is necessary to massage and mobilize the fracture daily without paying any attention to reduction which is often impossible to maintain. A proper reduction of the fractures cannot be made by any form of dressing and any apparatus is dangerous for a proper function of the joint. . . . If a vicious consolidation results in spite of proper treatment and there is a disturbance in the function of the forearm, the surgeon should not hesitate to operate and perform an osteotomy of the neck of the radius. The only inconvenience that may result from this operation consists in the appearance of a slight degree of cubitus valgus at the end of a certain time.

T. Turner Thomas (33), published an article entitled "Experimental Study and Report of Cases on Fractures of the Head of the Radius" in 1905. In the 45 patients reported, there were 48 fractures and only 1 of the patients was 13 years old, none below this age. Therefore, the subject of this paper was given relatively little consideration in the classical presentation of this subject by Thomas. He stated:

Instead of being an exceedingly rare fracture, as hitherto believed, it is a common one, its peculiarly obscure nature having rendered it especially difficult of recognition. There are more cases of this fracture, represented by skiagraphs in the hands of a few skiagraphers in Philadelphia (55), than the writers could find after a thorough search of the literature (48), making a total of 103. . . . When only the radial head is fractured, primary excision of the head should not be done. Excision of the head under any circumstances, even excision of the detached fragment, will rarely be required.

Union of the fragments will occur in many cases, even if moderate movements of the elbow be encouraged during the healing process. The possibility of non-union, however, will always be present, and as it will be difficult to determine the exact condition prevailing within the joint, it will probably be best in all cases to permit no movement for the first three or four weeks. Movements during this period probably do little good and may do much harm.

In 1907 Thomas (34) published another paper on "Fractures of the Head and Neck of the Radius" in which he discussed the problem of diagnosis; treatment was excluded.

In 1909 Hammond reported that he had seen 14 cases of fracture of the head of the radius in a total of 344 cases of all fractures. Relative to treatment he states:

In cases with good approximation of fragments and but slight displacement, I have had the best results with the right-angled tin splint, fashioned for the individual arm and supported by a sling. The splint should be left in place for several days to a week. It is then removed at intervals of two to three days and massage and gentle passive movements begun. . . . At the end of three to four weeks all apparatus may usually be omitted.

Where there is marked displacement of fragments which encroach upon the joint cavity, there will naturally be some ankylosis no matter what the treatment, and the question of operative interference arises. Each case must be judged on its own merits as shown by the roentgen examination, but the best results will usually be obtained when a conservative method of treatment has been followed, leaving the open operation for those cases of displaced fragment with limited motion in the joint, or the rare cases of non-union. If the plate shows a fragment so greatly displaced, that if left in position it will result in partial ankylosis, or is in danger of becoming a foreign body through non-union, it had best be removed by operation.

Bardenheuer states that he has never found it necessary to operate on a case of this fracture.

In 1910 Rabourdin stated:

With the exception of fissures with slight or no separation of fragments, it can be stated that the prognosis of these fractures is not very good from a functional point of view. . . .

Many different forms of treatment have been used. Some cases have been treated by massage and early immobilization and still others by an open operation. Seven of the patients among the 37 on which this thesis is based were treated by massage and early mobilization. In 5 of these cases there was a limitation of movement. Immobilization was used in 4 of these cases and satisfactory results were obtained.

An open operation was performed either early or late in 7 cases. Secondary resections were made in 5 other cases.

Many authors favor early mobilization with or without massage. However, an incomplete ankylosis occurs in many cases in which mobilization has been too hasty.

Immobilization has the advantage of relieving pain but should not be used in all cases. Good results are obtained in cases in which there is a simple fissure without any great separation of the fragments and without much deformation. However, mobilization should not be used in all varieties of this fracture. It is not possible to obtain a perfect position of the fragments if an apparatus of any kind is used.

An open operation may be performed immediately or when other methods of treatment have failed. A secondary operation usually gives good results and it has the advantage of being simpler than an early operation because the tissues are not contused or infiltrated with blood. However, in certain cases in which there is a great displacement of the fragments, an immediate operation is the method of choice. It has the advantage of acting on the fracture itself and also on the articulation. The operation consists in the removal of fragments and splinters. In most cases the fragments are too large and too mobile to be fixed in place. The elbow joint should be opened and any blood should be removed. An early operation diminishes the temporary incapacity and may prevent later complications.

. . . It is absolutely necessary to extract all splinters and perfect hæmostasis should be obtained. . . . The arm should be immobilized in a sling for about a week and then careful mobilization started.

Two years later (1912) Hitzrot (15) reported 29 cases of fracture of the head and neck of the radius. Of these, 19 involved the head and 10 the neck.

In 15 cases without displacement the arm was supported by a posterior splint, baking begun on or before the third day and massage on the fifth day. Movements in

flexion and extension were begun as soon as the swelling subsided, usually about the tenth day. Pronation and supination were begun about the fifteenth day. Result: Flexion and extension were complete in all cases. In 2 cases (chipping off the radial side of the head) pronation and supination were so slightly altered as to be classed as perfect. In 11 cases pronation and supination were half that on the normal side. In 2 of the same type as the first two cases mentioned, but with a line of fracture beginning well to the ulnar side of the articular surface of the head and extending outward through the head, supination was one-quarter and pronation one-half normal. Both the latter cases have some pain during supination and pronation.

In the 4 cases with displacement of the fragments the X-ray was necessary for the diagnosis of the displacement. In 2 the head was broken into three fragments, two of which were displaced anteriorly while one remained in position. In this type the entire head was removed by cutting through the neck about one-half inch below the articular surface of the head and removing the head.

In the 2 remaining cases the head was broken in one place only and the fragment which was radially placed was displaced outward and forward, and in this type the fragment only was removed.

The after-treatment was similar in other respects to that pursued in the fractures without displacement.

Results: The resulting motion was better in those cases in which the head was removed (i.e., the neck cut through at the lowest possible level). In the case last mentioned supination was present to seven-eighths the normal and pronation was complete. In the other cases of complete removal, supination was two-thirds normal and pronation seven-eighths normal.

In the 2 cases of partial removal of the head, supination was one-half normal, pronation two-thirds normal, and pain was present during either movement in one of the cases for nearly a year.

The 10 cases of fracture of the neck were all transverse in type and involved that portion of the shaft about one-half inch below the head. The X-ray was used to confirm the location of the fracture, and in none of these cases was there any marked displacement.

Treatment: With the arm in mid-position between pronation and supination a U-shaped plaster splint was applied and left on for from 3 to 5 weeks. Massage was begun on the tenth day and passive motion and rotation at the end of the third week.

Results: Pronation was complete in 2 cases, and in these 2 supination was also nearly complete. In the 8 other cases, pronation was two-thirds normal and supination five-eighths normal.

In 1914 Darrach expressed the opinion that when loose fragments involve a considerable portion of the internal aspect of the head of the radius, it is wiser to remove the whole head as such. Otherwise, the changes at the superior radio-ulnar joint would materially interfere with pronation and supination.

Jones (18) stated in 1915:

Fractures of the neck of the radius may occur with or without dislocation of the head of the radius forwards on to the front of the capitellum, or external condyle.

Treatment: (a) For the simple fracture of the neck the treatment is full flexion of the elbow with the forearm supinated. (b) When the head is dislocated as well as broken off, the displaced head may impede free flexion. The *simplest* treatment is to remove it. The lump of callus which forms around the broken end of the neck forms an excellent head under the molding forces of the ordinary movements of the part.

Fracture of head of radius. Fracture of a portion of the head is not uncommon. It is very apt to interfere with supination.

Treatment: The rule is to manipulate until supination is easily attained. If failure results then the loose piece can be removed. One of the chief causes of "clicking" elbow is malunion of a small portion of the fractured head of the radius.

As late as 1920, Lequerriere and Delhern believed that "Les fractures isolées de la cupule radiale sont très rares. Turner en 1905 n'en avait colligé que 48 cas en tout." Since they gave no other reference and must have meant T. Turner Thomas of Philadelphia one wonders whether this excellent monograph was available for their use.

Hitzrot in 1920 (16) called attention to the possibility that the "subluxation of the head of the radius" described as a lesion in children under three years of age (Stimson, and Jones and Lovett) "may, in certain cases be a separation of the epiphysis not readily detected even by X-ray." This author made the same division as Tanton:

1. Fractures of the head of the radius. These include all which occur in the region of the upper radio-ulnar joint capsule and include the epiphyseal separations.
2. Fractures of the neck of the radius. These include only those fractures that involve the bone below the region of the joint capsule and above the attachment of the biceps tendon.

The present authors agree with this division as previously stated.

Grossman (1923) stated that where a fragment has been separated and displaced, so as to block flexion of the forearm and it cannot be reduced by conservative measures, operative interference may become necessary whereby the offending fragment is either replaced in its proper position or removed. Storck (1924) believed that the demonstration of a loose fragment forms an indication for a surgical removal. Driberg (1924) stated:

In those cases in which the head of the radius is displaced, so as to cause mechanical obstruction, it is advisable to excise the displacement fragment without delay, and to start massage, etc., immediately after operation. If operation is postponed, there is liability of osteoarthritis and other joint and bony changes developing.

In the "mushroom" type of fracture, massage, movements, and exercises are usually sufficient to bring about a good result, but in some cases the flattening of the head of the radius causes considerable thickening and excess callus formation. If, therefore, at the end of a fortnight's treatment there is still decided limitation of flexion and

supination, I think it is advisable to operate and remove the head of the radius, taking care to get a full range of movements while the patient is under the anaesthetic.

Kellogg Speed, in 1924 (28), cites the detrimental results of excision but does not mention replacements. He says:

In children with growing bones and in infants, resection is not indicated. One must not interfere with the epiphysis for fear of growth deformity, and the loss of function is naturally overcome in great degree. (Length of time required is not stated.) Exceptions lie in ankylosed joints or those in which there is tendency to much new bone formation. After such resection in such children or adults, we use a modified arthroplasty by means of transplanted fascia.

Speed (27) again expressed his opinion in June, 1924 as follows:

Non-operative treatment is indicated in adolescents and children with fractures of the neck of the radius, even if decapitation of the bone has resulted from fracture. By direct pressure the loosened fragment may be forced into something approaching a normal relation with the rest of the bone and the forearm can be swung up and splinted in a position of complete flexion. After 12 or 14 days, active motions are begun with an especial attempt to re-establish pronation and supination. Baking, massage, and electrical treatments are strongly indicated. A happy functional result, with some loss of range of motion, may be anticipated in most cases. (Percentage not given.)

Sever (1925) discusses treatment without differentiation between children and adults. He states:

The relation of the articular surface of the head (radial) to the ulna and to the capitellum does not seem to be of great importance, except as originally laid down, to a good functional arm. However, if such a condition is found at once, i.e., an impaction with considerable displacement laterally and anteriorly of the head of the radius, no harm can result in attempting by an operation to replace (agreement with Grossman, 1923) the head in its proper position on the end of the radial shaft, and I believe that in most cases this should be done so as to restore as much as possible normal anatomic relations.

The fractures of the head which show in the roentgenograms that there has been a piece of bone chipped off, or show a marked alteration of the plane of the joint surface, generally require, at least theoretically, an operation to remove the loose fragment that acts as a foreign body in the elbow joint. This loose piece may also block free joint motion unless it is removed.

Fracture dislocations of the head of the radius, when there has been a complete separation of the head and neck with displacement of the head, should be operated on and the head removed. This is not generally enough to do, however; but to secure free motion in supination and pronation, the neck of the radius should be removed nearly to the bicipital tuberosity, after splitting of the orbicular ligament. The danger of the radius riding forward from the pull of the biceps, as the result of the escape of the neck from the orbicular ligament, is not of sufficient importance to weigh against free supination and pronation, which is often as limited after incomplete removal of the head, when the neck has been left, as before operation.

The more complete removal of the radius, following removal of the fractured head, will probably give a better end-result. This procedure, however, is not indicated as routine in all cases, but only in those necessitating a complete removal of the head.

Fairbank (1925) briefly states:

In fractures of the head and neck of the radius, if the displacement is not great, the elbow should be fixed in full flexion and the question of operation should be postponed till later. In many no operation is necessary. If, however, the whole or part of the head of the radius is grossly displaced, it should be removed forthwith.

C. W. Cutler, Jr., (1926) presented an excellent summary of the results of conservative treatment and excision of the radial head. He states:

Upon the basis of these results it would be manifestly impossible to make a satisfactory comparison between the operative and non-operative methods of treating fractures of the head and neck of the radius. The figures do indicate, however, that the closed method of treating simple cracks of the radial head produces satisfactory results. As regards fractures of the three other classes—(1) separation of one fragment; (2) fragmentation; (3) fracture of the neck—it can only be said that each method has yielded some results that were good, as well as a few that were imperfect. It would seem, therefore, unwise to advocate excision in every case of fragmentation or fractured neck. This is especially true since the operative procedure itself is not free from danger. The technical difficulty of locating and removing a single displaced piece of the head, or of finding and extracting all pieces in a multiple fragmentation, may be considerable. In one case of this group, at least, not all of the fragments could be extracted. In addition three of the patients operated upon suffered infection of the wounds, resulting in delayed convalescence and in impaired results in two. Considering also the fact that should the closed method fail of good results in appropriate cases recourse may still be had to surgical removal of the fragments, it would seem best to treat these injuries without operation except where definite indications for removal are present. Such indications would appear to be: (1) such displacement of a fragment or of the whole head as would interfere with full joint motion; (2) irreducible complicating dislocation of the radius or ulna or both; (3) malunion, ankylosis or impaired motion in old cases.

DeWaard (1926) is positive relative to indications for excision. He says:

When the capitellum of the radius is completely detached, as well as displaced, it must be operatively removed, and the end-result is then not quite so favorable as on conservative treatment of simple fractures of the radial head. When the radial capitellum is not essentially displaced, it can be restored to its normal position by operative procedures. This portion of the elbow joint is not to be removed without urgent necessity. The good end-results obtained in children who were conservatively treated, indicate that this mode of treatment is here entitled to preference, unless a strong displacement of the fragments require an operative intervention. In view of a good future function, the preservation of the radial head is indicated in operative interventions. The suggestion has been made, but it is not justified, to remove in case of detachment of a piece of the radial head, not only

this fragment, but the entire head of the radius. When the radial head is broken off as a whole, without more than a displacement being demonstrable in the roentgen picture, a replacement of the radial head in its proper position by operative measures is called for (agreement with Grossman, 1923, Sever, 1925). The head of the radius is too important a portion of the elbow joint to justify its removal without urgent reasons, such as a comminuted fracture, with shattering and splintering of the bone.

Pfab (1929) reported 50 cases seen between 1922 and 1928. Twenty-five were treated by the closed and the other 25, by the open reduction method.

Fissures, separations, and fractures without dislocation must be treated conservatively by means of an elastic bandage, hot air baths, and massage. The results are good. Dislocated fractures must be treated surgically; the reduction and fixation of the head gives better results than extirpation (agreement with Grossman, 1923, Sever, 1925, DeWaard, 1926); the latter is the method of choice in compound fractures. The reposition gives excellent results. The extirpation is usually followed by some limitation of motion, especially in the sense of pronation and supination, and an occasional appearance of free joint bodies. The postoperative complications can be prevented to a great extent by padding of the radial stump with fascia, obtained by a free transplant from the fascia lata.

Key, 1931, states:

In children, if the displacement is slight, the fracture should be treated conservatively with the expectation that growth will correct the slight deformity. If the displacement is marked, open operation is indicated and this should be done as soon as practicable after the injury; but the head of the radius should not be removed as is recommended in adults for the same type of fracture (agreement with Grossman, 1923, Sever, 1925, DeWaard, (32) 1926, Pfab, 1929).

In children the head can be manipulated or picked out and put back in its normal position on the neck and held in position by placing one or two sutures of fine catgut in the periosteum of the neck and then suturing the annular ligament around it. In this operation it is important that the elbow and forearm be placed in a position in which the head is fairly stable on the neck (a flexion of 45 degrees in a midposition between pronation and supination in my cases). After the head is placed in position, the forearm should not be moved until the wound is closed and the posterior plaster mold has hardened. This is of great importance, so slight movement may not displace the head, which is fixed very insecurely on the neck.

Within the past year I have replaced the head in 2 cases in children and both now have approximately normal motion in flexion, extension, pronation, and supination, normal power in the forearm and no pain. I have not attempted to replace it in adults.

The only contribution I feel I have made is the replacement of the head in children. In the literature I have not found that this has been done.

From the chronological review above given it is evident that the advisability of replacing the radial head and neck to correct malalignment has been advocated by an increasing number of authors since 1923.

CASE REPORTS

CASE 1. B. L., S. M. H. No. 62879, aged 11 years, white, schoolgirl, entered emergency division Strong Memorial Hospital, July 8, 1932. Twelve hours previously the patient tripped and fell on the floor on her extended and pronated left forearm. There was immediate pain and inability to use the elbow. The patient was brought to the hospital because, after 12 hours, the pain had still not abated. The past history was irrelevant. She had never previously broken any bones. General physical examination showed a well developed and nourished girl of 11 years. The various systems were negative. *The left elbow* was held in 90 degrees' flexion with the forearm midway between pronation and supination. The forearm was supported by the right hand. There was moderate swelling about the elbow joint. Passive flexion and extension of the elbow were possible to 90 degrees and 145 degrees, respectively, without discomfort. Only a few degrees of passive supination were possible because of pain. *There was marked pain on gentle pressure over the head of the radius with referred pain to the distal end of the radius.* No abnormality in contour of the radial head could be made out. Roentgenograms of the left elbow showed a fracture of the left radius just below the proximal epiphysis with definite lateral and anterior displacement of the proximal fragment (Fig. 1).

Laboratory findings: Hæmoglobin, 80 per cent; white blood cells, 7,800; urine, clear, yellow, acid, specific gravity, 1.010; albumin, negative; sugar, negative; microscopic, few white blood cells; Wassermann reaction, negative.

Treatment. An open reduction was done at once under ether anæsthesia. A linear incision was made on the lateral side of the forearm with its midpoint over the radial head. An impacted fracture of the radial neck was found. This had resulted in a displacement, anteriorly and laterally, of the radial head. The deformity was corrected by leverage with a periosteal elevator at the line of fracture (Fig. 2). The impaction gave stability to the fragments. No internal fixation was used. The periosteum was closed with interrupted silk sutures. The remainder of the wound was closed in layers with the same suture material. The elbow joint was immobilized in 90 degrees' flexion and full supination with a posterior plaster splint. The wound healed *per primam.*

The splint was removed at the end of 3 weeks. Physical therapy by means of baking, massage, and gentle passive motion, was commenced.

Seven weeks after operation the patient had a perfectly normal elbow joint (Figs. 3 to 6).

CASE 2. P. L., S. M. H. No. 65243, aged 7 years, white schoolgirl, entered the Strong Memorial Hospital emergency division, September 15, 1932. Patient fell one hour before admission. The fall had been stayed by the pronated right hand, the elbow being in complete extension. The past history was irrelevant. She had never broken any bones previously. General physical examination showed a well developed and nourished girl of 7 years. The various systems were normal with the exception of the right elbow. *The right elbow joint* was held in 90 degrees' flexion with the forearm in complete pronation. There was moderate swelling of the region with ecchymosis. On palpation there was an abnormal prominence in the region of the radial head and on gentle pressure over this area the patient winced with pain. Passive flexion was possible to 70 degrees and extension to 160 degrees without undue discomfort. Attempts at passive supination were painful and markedly limited. The patient complained of referred pain over the distal end of the radius as much as that produced by pressure of the index finger applied over the radial head and neck.

Laboratory findings were normal: hæmoglobin, 91 per cent; white blood cells, 8,400; urine, clear, yellow, alkaline; specific gravity, 1.005; albumin, negative; sugar, negative. The sediment showed an occasional white blood cell. Wassermann reaction was negative.

Roentgenograms revealed a fracture of the radius just below the proximal epiphysis, with lateral and anterior displacement of the radial head (Fig. 7).

Treatment. On entry a closed reduction was attempted. This was not successful. Eight hours later an open reduction was done under ether anæsthesia. A 3 inch curved incision with its midpoint over the head of the radius and convexity toward the olecranon was made on the lateral side of the forearm. The head of the radius was found to be broken off completely and displaced anteriorly and laterally. It was replaced by gentle leverage with a periosteal elevator between the fragments. The fragments remained in apposition even when the forearm was pronated or supinated or the elbow flexed or extended (Fig. 8). No internal fixation was used. The annular ligament was repaired and the wound closed in layers with silk. A posterior plaster splint was applied to immobilize the forearm in 90 degrees' flexion and full supination. The wound healed *per primam*. The patient was discharged from the hospital on the eighth day after operation. Immobilization was continued for 4 weeks. Splint was then removed and the patient encouraged to use the arm. No physiotherapy was used.

Two months after the operation the patient had complete range of motion in the radiohumeral joint (Figs. 9 to 12). Roentgenograms showed healing at the level of fracture.

Case 3. J. B., S. M. H. No. 68235, aged 11 years, white, schoolboy, entered Orthopedic OPD of Strong Memorial Hospital, November 29, 1932. Two hours before entry the patient had been shoved out of a chair by another schoolboy. He fell upon his flexed left elbow. There was immediate pain in the elbow on attempts at motion. The past history was irrelevant. He had never sustained any fractures previously. Physical examination showed a well developed and nourished boy of eleven years. The various systems were normal. *The left elbow* revealed slight swelling. The forearm was supported by the right hand. The elbow was held in 90 degrees' flexion; the forearm midway between pronation and supination. Passive flexion and extension were limited by pain to 90 and 120 degrees, respectively. Passive supination was possible through about 25 per cent normal range. It was accompanied by definite crepitus over the radial head and caused pain referred to the region of the radial head and to the distal end of the radius. Gentle pressure over the radial head elicited no crepitus but caused referred pain to the two regions mentioned. No abnormality in contour of the radial head could be palpated.

Roentgenograms showed a separation of the proximal epiphysis of the left radius, displaced into the joint as if hinged posteriorly to the radius. There was an associated hair line fracture without displacement through the lateral epicondyle of the humerus (Fig. 13).

Laboratory findings: hæmoglobin, 76 per cent; white blood cells, 8,450. Urine, cloudy, yellow, specific gravity, 1.014, alkaline; albumin, negative; sugar, negative; microscopic, occasional epithelial and white blood cells. Wassermann reaction was negative.

Treatment. The patient was admitted to the hospital on the following day and an open reduction was done under nitrous oxide gas-oxygen-ether anæsthesia. A linear incision, 3½ inches long, was made on the lateral side of the elbow with its midpoint over the radiohumeral joint. The joint was opened. On doing so there was an escape of 25 to 30 cubic centimeters of thick dark blood. The epiphysis was found displaced far posteriorly and completely separated from the radial shaft except for a small posterior hinge. With some difficulty it was replaced in normal relationships with the radial neck. It was found to be quite unstable except when the forearm was placed in supination and the elbow in complete extension. This kept the epiphysis wedged between the radial neck and the capitellum of the humerus (Fig. 14). The capsule of the joint was closed with interrupted silk sutures and the wound, in layers with the same suture material. The elbow joint was immobilized in complete extension and the forearm in supination, with a posterior plaster splint. The wound healed *per primam*. One week after operation the elbow joint was gently flexed to 90 degrees without anæsthesia and a posterior plaster splint was applied. Roentgenograms following this revealed that the epiphysis had remained in normal position. Three weeks after operation this splint was removed and the patient encouraged to use the arm. No physiotherapy was used.

Two and one-half months following injury the patient had complete flexion in the elbow, extension, 175 degrees; supination, 90 per cent; and pronation, normal (Figs. 15 to 18). The patient had no symptoms, on motion crepitus could be felt in the joint. The X-ray negatives at this time showed the epiphysis in normal position. There were several minute bone fragments in the joint interpreted as being due to small fragments broken off at the original injury.

SUMMARY

Restoration of function in groups A and B with relation to treatment given:

I. Group A. Fractures within the radiohumeral joint.
  A. Fractures of the radial head.
    1. Linear and chip fractures with little or no displacement.
      a. Treatment: Immobilization at 90 degrees' flexion with full supination 14 to 28 days, followed by massage.
        (1) Result: All motions normal or only slight limitation in one or more.
    2. Linear and comminuted fractures with displacement of one or more fragments.
      a. Treatment: See A, 1.
        (1) Result: Moderate to severe limitation in pronation and supination, usually involving flexion and extension.
          (a) Can be improved only by late resection of radial head and neck.
      b. Treatment: By immediate operative correction of deformity. Three weeks immobilization as above. Baking and massage.
        (1) Result: Minimum to moderate limitation in one or more motions. Probable prevention of resection of radial head and neck.
      c. Treatment: If immediate operation reveals that deformity cannot be corrected, then radial head and neck should be resected.
    3. Displacement of radial epiphysis.
      a. Treatment: Immediate operative correction. Two to 4 weeks' immobilization.

May or may not be followed by physiotherapy.
(1) Result: Little or no limitation in pronation and supination. Flexion and extension normal.

b. Treatment: Conservative treatment always followed by definite impairment of function in two or more directions. Frequently requires resection of radial head and neck.

4. Linear fractures from radial head into neck.
a. Treatment: See A, 1 and A, 2.
(1) Result: See A, 1 and A, 2.

II. Group B. Fractures outside the radiohumeral joint.
A. Fractures of the radial neck between the level of the synovial membrane attachment and the insertion of the biceps tendon.
1. With minimum or no malalignment.
a. Treatment: Immobilization, 90 degrees' flexion and full supination, 2 to 4 weeks, followed by baking and massage.
(1) Result: Little or no limitation of function in any motion of radiohumeral joint.
2. With definite malalignment.
a. Treatment: Operative correction of deformity. Immobilization, 90 degrees' flexion, full supination 2 to 3 weeks, may or may not be followed by massage.
(1) Result: Little or no limitation in pronation and supination. Flexion and extension of the elbow normal.
b. Treatment: Conservative.
(1) Result: Invariably leads to disability which can be improved only by resection of radial head and neck.

III. Group C. It is obvious that the variations mentioned are included in the summary of groups A and B.

CONCLUSIONS

Traumatic swelling on the lateral aspect of the elbow joint should direct attention to examination of the head and neck of the radius.

Intra-articular or extra-articular relationships which are detrimental to the restoration of function of the radiohumeral joint cannot be favorably influenced by manipulation and fixation. Immediate mobilization has been found equally detrimental to the restoration of function of this articulation.

The limitation of function which invariably follows these generally employed conservative measures has led to the necessity of frequent resection of the radial head and neck.

The head and neck of the radius are essential for stability of the elbow joint and maintenance of the normal carrying angle of 10 degrees.

The removal of the radial head and neck may improve pronation and supination, but it is always followed by deformity in terms of an increased carrying angle, and relative instability of the elbow joint.

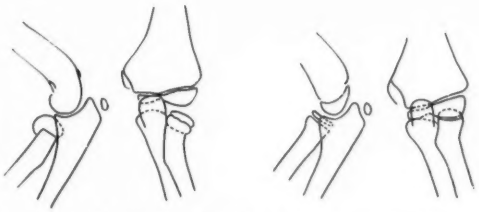

Fig. 1, left. Case 1. Tracing of roentgenograms of left elbow on admission. July 8, 1932.
Fig. 2. Case 1. Tracing of roentgenograms of left elbow after operation. July 9, 1932.

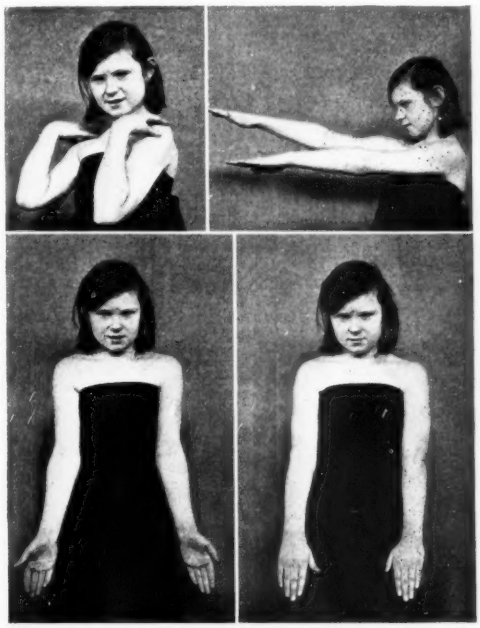

Fig. 3. Case 1. Flexion 5 months after operation.
Fig. 4. Case 1. Extension 5 months after operation.
Fig. 5. Case 1. Supination 5 months after operation.
Fig. 6. Case 1. Pronation 5 months after operation.

This paper is presented as additional evidence in support of the premise that immediate operation is indicated for the correction of (1) displaced radial epiphyses, (2) displaced fragments of the radial head (removal if necessary), and (3) malalignment of fragments in fracture of the radial neck.

Pressure with the index finger over the affected radial head and neck produces referred pain at the distal end of the radius. On the first observation of this clinical sign the severity of the pain directed the making of roentgenograms to deter-

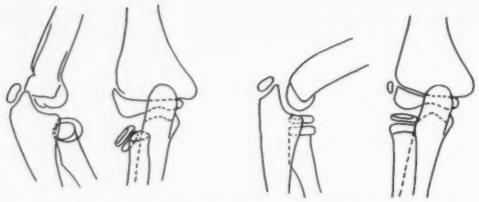

Fig. 7, left. Case 2. Tracing of roentgenograms of right elbow on admission. September 15, 1932.
Fig. 8. Case 2. Tracing of roentgenograms of right elbow after operation. October 14, 1932.

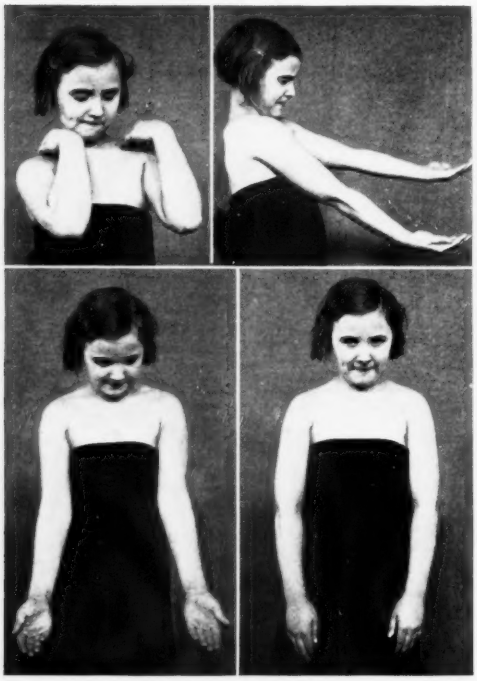

Fig. 9. Case 2. Flexion 3 months after operation.
Fig. 10. Case 2. Extension 3 months after operation.
Fig. 11. Case 2. Supination 3 months after operation.
Fig. 12. Case 2. Pronation 3 months after operation.

mine the presence or absence of injury to the distal third of the radius. This observation has been made in 5 successive patients, including the 3 which have been reported. We have found no report of this clinical sign in the 34 references quoted.

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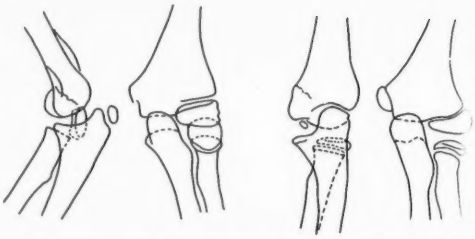

Fig. 13, left. Case 3. Tracing of roentgenograms of l t elbow on admission. November 29, 1932.
Fig. 14. Case 3. Tracing of roentgenograms of l t elbow after operation. December 3, 1932.

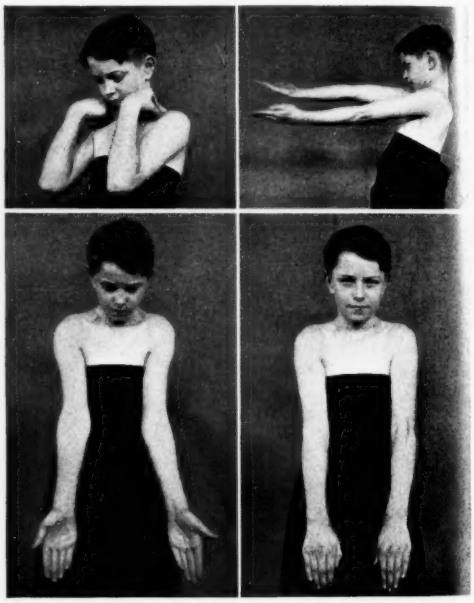

Fig. 15. Case 3. Flexion 2½ months after operation.
Fig. 16. Case 3. Extension 2½ months after operation.
Fig. 17. Case 3. Supination 2½ months after operation.
Fig. 18. Case 3. Pronation 2½ months after operation.

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NEPHROPEXY

Present Day Status and Description of a New Technique[1]

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At the present time urologists of many different parts of the world have returned to the beneficent operation now known as nephropexy. Indeed, no operation has a more fascinating history. Its discovery was foreshadowed when Rayer noted that relief from the symptoms of movable kidney could be obtained by rest in bed and by mechanical support in the form of a pad or belt. Beginning with this observation surgeons began to make definite efforts to relieve this particular condition by surgical suspension of the kidney, assuring its permanent fixation in the proper position. However, due to mistaken choice of the operation for conditions not warranting its use in any way, surgeons in general came for quite a period to cast doubt on the value of the operation and succeeded in discrediting its value. However, the operation has survived.

The first attempt to secure a movable kidney was that of Dr. Greensville Dowell, of Galveston, Texas, who, in 1875, attempted to secure the kidney by means of a seton. In 1870, Gilmore, another American surgeon, successfully removed a painful, atrophied, floating kidney from a 5 months' pregnant woman by the lumbar region; the second successful deliberate nephrectomy in history and the first surgical intervention on the kidney during pregnancy. In 1878 Martin popularized nephrectomy for the relief of movable kidney. Radical removal of the kidney gained some favor because it was an intervention that permanently cured the patient. It soon became apparent, however, that this radical operation sacrificed a relatively healthy kidney, could not be performed when the opposite kidney was damaged or involved by stone formation and when renal mobility was bilateral. With these facts in mind, Hahn, in 1881, conceived the idea of fixing the kidney by sewing the perirenal fat to the walls of the lumbar incision by suture. He called this operation nephrorrhaphy. Eight years later Le Dentu wrote his excellent work entitled "Affections du Rein et des Uretères" in which he devoted an entire chapter to surgical suspension of the kidney and in which he stated that the term nephrorrhaphy led to confusion as it signified suture of the kidney itself. For fixation of the mobile kidney to the posterior abdominal wall by suture he introduced the term "nephropexy" which came from the Greek meaning to fix and to coagulate. This term was soon adopted and has been universally employed since that time. Hahn's original operation consisted of suturing the fatty capsule to the muscles of the lumbar incision. Although this operation did not give lasting results, it paved the way to the perfection of various types of operations that permanently relieved the patient. Surgeons soon began lending their efforts to the invention of numerous and ingenious methods of nephropexy. Many of these were never adopted or were soon given up for methods that afforded the highest percentage of lasting fixation. After the operation had been perfected so that the percentage of failures had been reduced to a minimum, surgical suspension became exceedingly popular. Some surgeons eager to follow the new fashion, little heeded the criteria for operation advised by Albarrán and many other sane thinking urologists, namely pain, obstructive phenomena and gastro-intestinal symptoms, and began fixing movable kidneys that were causing no symptoms. Thus, then, nephropexy soon became the finishing touch, the master stroke of nearly every abdominal operation: The result—and a quite logical result—was that in many instances the operation gave little relief because it had been depended upon to relieve renal obstruction that did not exist. But this experience at once led numerous surgeons to give up the operation entirely. And thus the pendulum swung far and wide to such an extent that the patient suffering from severe attacks of Dietl's crisis due to strangulation of a movable kidney had difficulty in obtaining this due surgical relief. Unfortunately, nephropexy is still held in disrepute by certain surgeons and urologists, who, in recent publications, emphasize the failure of this operation in cases in which as usual it had been wrongly applied, and these surgeons, either willfully or blindly, fail to recognize its unquestionable beneficent results in the numerous cases to which it is being intelligently and more correctly applied.

At the present time most of the surgeons performing nephropexy fix the kidney by means of

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utures taken through its substance or fibrous apsule or secure it by suture of the partially reected fibrous capsule. Some surgeons employ ands of muscle, fascia, or tendon secured from he surrounding structures in order to perform ixation. Suspension by support from below obained by closure of the renal fossa by approximaion of the perirenal fascia, fatty capsule and eritoneum, also has its advocates. Others emloy the combined method of fixation in which hey fix the kidney by sutures passed through the apsule, the parenchyma (Papin) or partially reected fibrous capsule, and secure additional support from below by massing the renal fascia and eritoneum.

At the present time there seems to be some confusion as to the choice of the operative procedure to be employed in the relief of movable kidney presenting symptoms, and these symptoms, it may be remembered, are pain, gastro-intestinal disturbances, nervous phenomena and the persistent infection of the kidney resulting from faulty drainage. Very recently some surgeons, notably Hess, Herbst, and others, claim that denervation or renal sympathectomy, alone, will relieve the patient of the symptoms of movable kidney. While this procedure unquestionably relieves the pain associated with this condition, it should be reserved for those cases of nephralgia in which faulty drainage due to stasis plays a minor rôle, if any. Nephropexy is the operation of choice in patients in whom movable kidney is causing marked symptoms, certainly in those in which the lowered position of this organ causes faulty drainage and persistent infection of the kidney, and in those who cannot tolerate abdominal belts and mechanical supports.

The development of urography by means of which renal ptosis, ureteral angulation and back pressure changes in the kidney can be actually demonstrated on the X-ray film afforded the clean cut scientific evidence necessary to point out the true indications for this operation with the result that surgeons no longer need be guided by misleading palpatory evidence. And furthermore, the quite recent intensive work on the ureter has revived and has again justified this beneficent operation which was almost lost to the profession.

At the present time urologists of many different countries of the world have added their guiding influence in the development of nephropexy. In England we find Bell; in France, Marion, Chevassu, Papin, Heitz-Boyer, and Legueu; in Germany, von Lichtenberg; in Spain, Sánchez Covisa, Cifuentes, and Miraved; in Cuba, Ledón, and in our own United States, the late B. A. Thomas, Bransford Lewis, Peacock, Fowler, Lowsley, Bissel, Burford, Hinman, Scholl, Squier, Deming, and Mathé. All of these men have added their bit to replace nephropexy on a sane and scientific basis and to make it take its proper importance among surgical interventions on the kidney.

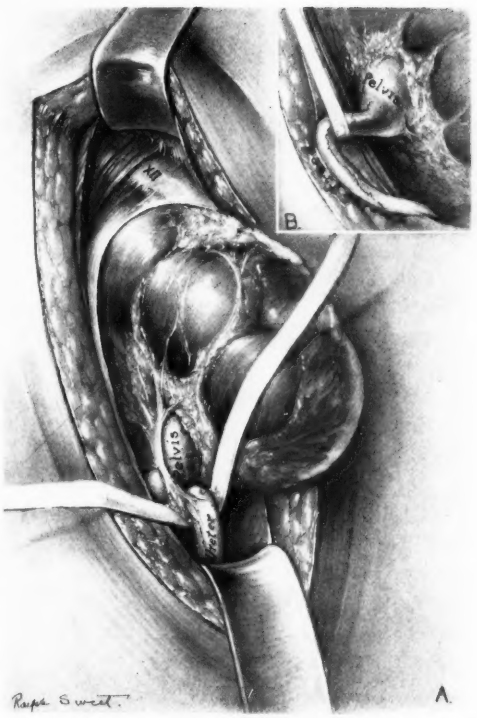


Fig. 1. First step: A, Entire kidney is freed and delivered into the incision. The upper ureter is freed (ureterolysis) in order to eliminate any pressure that might be exerted thereon by aberrant vessels or fibrous bands. B, Ureter dissected free.

But of course the ideal operation for surgical suspension of the kidney should be one that accomplishes fixation of the kidney in the renal fossa in a sufficiently high position by which free drainage from the kidney is assured and by which all kinks in the ureter can be obliterated. In rechecking various methods in vogue for this purpose the author observed that in some instances the kidney had not been fixed in a satisfactorily high position to make possible the best drainage and freedom from kinks in the ureter. This led the author to devise a new method for the regulation of the height of suspension of the kidney,

Fig. 2. Second step: Illustrates denervation or renal sympathectomy. The sympathetic nerve fibers are easily located on the superior surface of the renal artery or its main branches where they are severed. Exposure is facilitated by retracting the vein with a small retractor.

and by which one could replace this organ in an ideal anatomical situation, assuring perfect drainage and lasting relief of symptoms. This method consists of fixation of the organ high up in the renal fossa by taking sutures in the fibrous capsule of the kidney and attaching them above the eleventh or twelfth rib.

Before describing this method it is well, in order that the need for a new and more satisfactory scheme of suspension may be fully understood, to review the methods of surgical suspension by means of the fibrous capsule which have formerly been employed. It is interesting that all through these various and very similar methods it is possible to follow the fascinating history of an idea as it proceeds little by little to its present form as illustrated in the "present-day technique and status of nephropexy."

Bassini, 1881, was the first surgeon to fix the kidney by means of its capsule. In the same year Robert Weir, of New York, independently carried out a similar technique to that of Bassini in performing the first recorded nephropexy in the United States. In 1888, Duret, of Lille, proposed his method of fixation by the fibrous capsule. These surgeons emphasized the fact that one should fix the kidney by passing sutures through the more resistant true capsule rather than through the rather loose fatty capsule. As a result of experimental work conducted by Bassini, 1888, Vanneufville, 1888 and Tuffier, 1889, in which it was definitely proved that fixation by means of sutures passed through the parenchyma of the kidney caused no lasting damage to this organ, surgeons soon began fixing the kidney by means of sutures passed through its substance. The

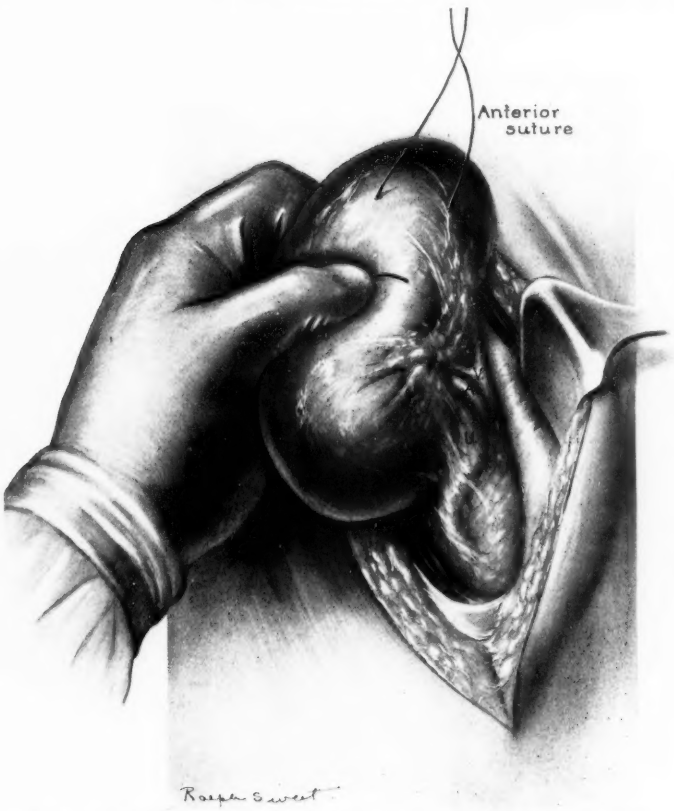


Fig. 3. Third step: A triangular twenty day chromic catgut suture is passed through the fibrous capsule on the anterior surface of the upper pole of the kidney leaving two outer bridges on the convex surface of the kidney.

method of suspension gained considerable favor and fixation by means of sutures passed through the capsule of the kidney was not revived until the beginning of the twentieth century, when Goelet, in 1902, described his method and declared that it was not necessary to pass sutures through the kidney substance nor was it wise to remove its fibrous capsule in order to secure fixation.

While Goelet was working on this method of securing the prolapsed kidney, Max Brödel of Baltimore, well known anatomical artist, devised his triangular suture later known as the Brödel stitch which was passed through the renal cortex and fixed to the quadratus lumborum muscle. Howard Kelly soon adopted Brödel's triangular suture of fixation by the fibrous capsule and was among the foremost kidney surgeons of the United States to point out the beneficent results obtained by performing nephropexy in indicated cases. His guiding influence and keen judgment has done much to place nephropexy on a firm basis in this country.

Although Kelly's method has given him and his numerous pupils excellent results, the author observed in checking his personal results from its use by pyelography that the kidney was not always fastened in a satisfactory position. Likewise the other methods heretofore described by which the kidney was fixed by sutures taken in its fibrous capsule gave excellent result in many cases, yet in some instances it did not replace the kidney high enough in its fossa nor in its normal anatomical position. These very uncertain ele-

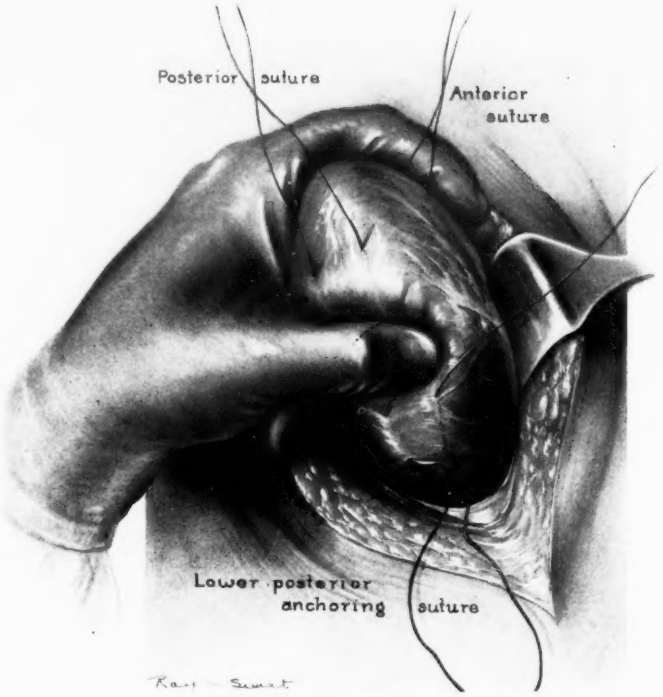


Fig. 4.

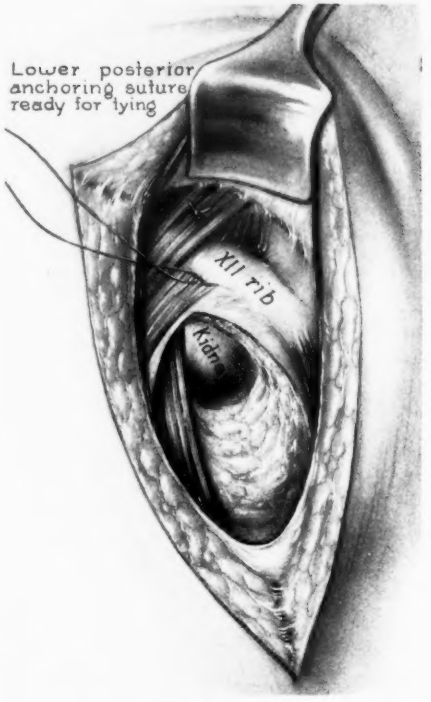
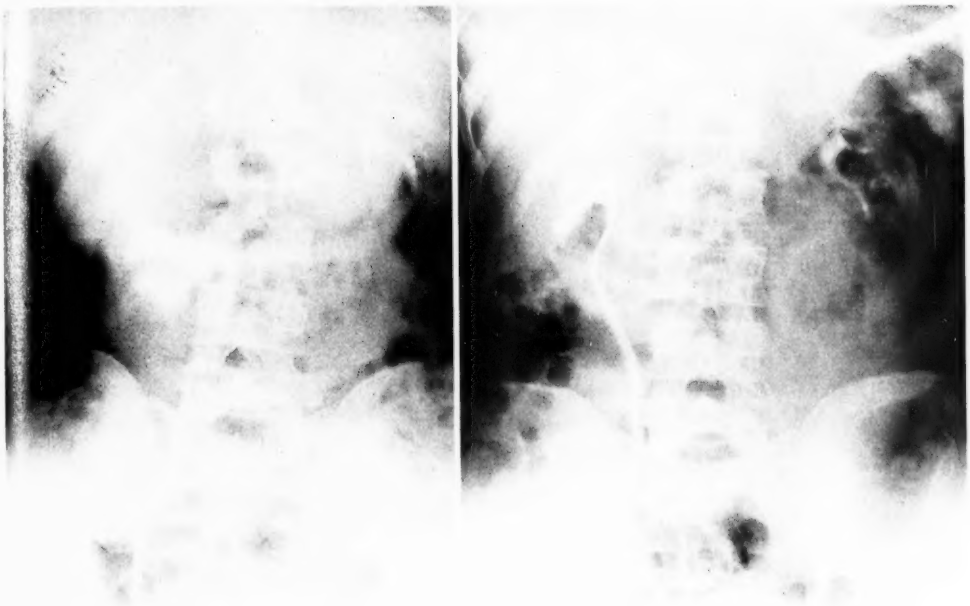


Fig. 5.

Fig. 4. Fourth step: Similar triangular sutures are taken on the posterior surface of the capsule in the region of the upper and lower poles of the kidney.

Fig. 5. Fifth step: Ligation of the upper anterior and posterior suture above the twelfth rib. In cases in which high suspension is desired, these are placed at a lower level on the kidney and are ligated above the twelfth rib. Note the ligation of the lower posterior steadying suture to musculature below the twelfth rib, which prevents torsion of lateral displacement of the kidney.



A. B.

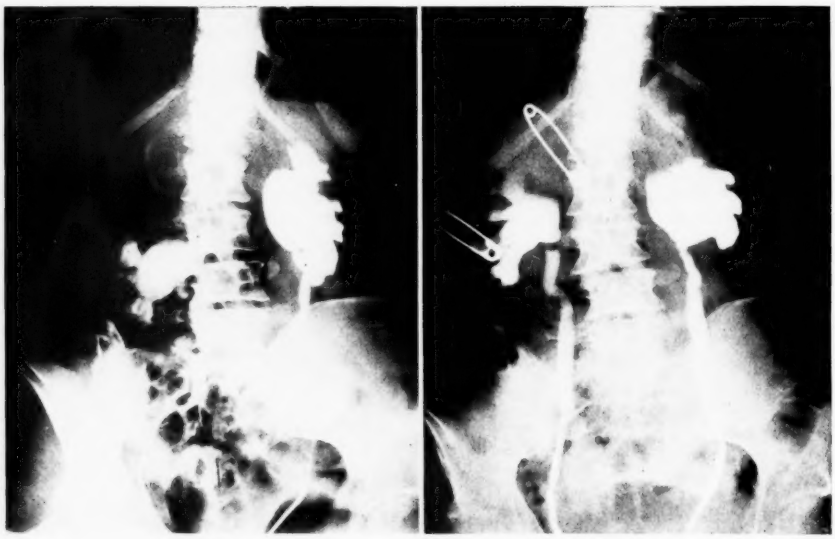
Fig. 6. A, Pyelogram taken of a patient aged 31 years, presenting second degree renal ptosis. In the pre-operative pyelogram one remarks the lowered position, torsion of the kidney, and early hydronephrosis. B, Pyelogram taken 6 months after the author's method of nephropexy. Note high fixation in the normal anatomical position by which the ureter has been straightened and good dependent drainage of the kidney assured.

ments of the old operations which led the author to seek to devise a more satisfactory operation, one by which the kidney can be fixed at any height desired, by which it is entirely freed from the surrounding structures and assuring that the upper portion of the ureter can be readily exposed. This last is to eliminate any back pressure on the tube from adhesive bands or aberrant vessels which left untouched might defeat the purpose of the operation.

As may be seen, the idea which threads through all the efforts of these many surgeons was the idea that a means could be discovered by which a satisfactorily high suspension could be achieved and maintained, a lasting fixation of the movable kidney in a position which would insure good drainage of urine and relief from symptoms. With this idea as a goal, the author perfected a technique which he recommends and presents in detail as a successful and beneficent method for satisfactory suspension and fixation of movable kidney.

AUTHOR'S METHOD OF NEPHROPEXY

The usual curvo-linear Albarrán incision is made extending from the costovertebral angle toward the anterior superior spine. The kidney is entirely freed from the surrounding structures, (nephrolysis) delivered into the incision and liberated from sclerizing fibrosis or fibrolipomatosis that might be present usually due to an accompanying perinephritis. A triangular 20-day absorbable chromic catgut suture is taken in the anterior and posterior surface of the kidney either in the region of the upper pole, the mid region, or in the junction of the mid region and lower pole depending on the height of fixation desired. The triangular suture is taken in the renal capsule in such a manner as to leave two outer bridges on the surface of the kidney and in the most advantageous position where it would counteract forces tending to bring the kidney to a lower level. The direction of the suture is made so that it is at right angles and not parallel to the framework of the cortex (Fig. 3). Suspension at a greater height



A. B.

Fig. 7. A, Female, aged 52 years, presenting marked gastro-intestinal symptoms, nervous disturbances, and repeated attacks of Dietl's crises. In the pre-operative pyelogram, taken in the dorsal position, one notes bilateral third degree ptosis with marked kinking of the ureter and hydronephrosis. B, Pyelogram taken after operation demonstrating satisfactory fixation of both kidneys in high position, unimpeded drainage of the kidney and regression of hydronephrosis.

can be obtained by placing the sutures lower down in the kidney. These sutures are more easily passed through the musculature above the twelfth rib by employing a Reverdin needle where they are ligated separately. In cases requiring very high suspension the upper anterior and posterior sutures are tied above the eleventh rib and in passing the Reverdin needle downward in order to draw up the fixation suture, it is well to keep close to the posterior surface of the anterior wall of the thorax in order to avoid injury to the pleura. In this way one can obtain high fixation of the kidney thereby taking up any slack that might exist in the ureter, assuring straightening of any kinks that might be present in this tube. A third suture is taken in the posterior surface of the kidney about 2 centimeters below the upper, posterior suture, and is anchored to the musculature below the twelfth rib in order to steady the kidney and in order to prevent torsion or lateral displacement of this organ. The upper portion of the ureter is routinely dissected free from the surrounding structures (ureterolysis) in order to eliminate any pressure that might be exerted thereon by aberrant vessels, fibrous bands, etc., which, left untouched, might defeat the purpose of the operation. In cases presenting an unusual amount of pain, denervation of renal sympathectomy is also performed. This consists of severing the sympathetic nerve fibers which are usually found to course along the superior surface of the renal artery and its main branches. Exposure of the renal artery is facilitated by retracting the renal vein with the use of a small retractor which is shown in Figure 2.

In cases in which there is a concomitant perinephritis in which the fibrous capsule consists of a thickened, indurated, sclerotic shell, partial decapsulation is performed in order to relieve strangulation of the kidney. A small longitudinal incision is made in the capsule on the anterior convex surface of the lower pole of the kidney. The capsule is partially stripped and this is sufficient to release the kidney and does not in any way interfere with the integrity of that portion of the renal capsule on which the fixation sutures are taken. A soft rubber tissue drain is placed against the posterior surface of the lower pole of the kidney and it is brought out through the upper portion of the skin incision. It is gradually withdrawn about a centimeter each day, in such a way that at the end of a week it is entirely removed. The patient is kept in bed in the Trendelenburg position for 3 weeks assuring

adhesion of the kidney to the walls of the renal fossa in a high position.

The results obtained from employing this method on numerous cases in St. Mary's Hospital, in the French Hospital, and in the Southern Pacific General Hospital in San Francisco have been very satisfactory. Routine postoperative pyelographic study has demonstrated that the kidney had been permanently fixed in a sufficiently high position by which the ureter had been freed of kinks and this good dependent drainage of urine was definitely established. Relief from symptoms by the employment of this method has surpassed the use of all other methods formerly employed by the author.

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EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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OCTOBER, 1933

THE PRESENT STATUS OF RADIUM THERAPY IN CANCER OF THE UTERUS

RADIUM therapy at the present time is being weighed in the scale of experience against surgery in the treatment of cancer of the uterus. In considering the value of a mode of treatment, it is essential that we differentiate clearly cancer of the cervix from cancer of the fundus. In about 10 per cent of cases, cancer originates in the cavity of the fundus uteri usually as adenocarcinoma. Cancer of the fundus is more frequent after the menopause, the mean age incidence being 8 years more than in cancer of the cervix.

The ordinary panhysterectomy gives an average 5 year cure rate of 60 per cent in these cases and is therefore the method of choice, but unfortunately on account of senility, obesity, cardiovascular disease, diabetes, many of these patients are poor surgical risks, so that we must resort to intra-uterine radiumtherapy and X-ray, which give a cure rate closely approaching surgery. Probably in these patients the ideal method is to combine radiationtherapy and surgery when possible.

In cancer of the cervix all authorities agree that surgery is of little avail unless the radical operation of the Wertheim or Schauta type is employed. This operation can be done only in cases classed as operable. For the inoperable class, which includes more than 50 per cent of those seeking relief, surgery has nothing to offer.

In competent hands radiumtherapy combined with high voltage X-ray in advanced cases gives results which are fully equal to the 5 year results obtained in surgery in the "operable" patients, the primary mortality rate being less than 2 per cent as compared to the 8 to 17 per cent in radical surgery. For the "inoperable cases" radiumtherapy gives an average 5 year cure rate of 12 to 18 per cent and has in the majority of cases a definite value in the palliation and prolongation of life.

A 5 year cure rate of approximately 25 per cent may be obtained in cancer of the cervix uteri by either radical surgery or radiumtherapy in all those applying for relief. In the early cases in which the disease is limited to the cervix, a 50 per cent or better 5 year cure rate may be expected.

The radical Wertheim operation requires skill and experience to be properly performed and it must not be confused with the usual operation of complete or panhysterectomy which is frequently done for carcinoma of the cervix. The difference between an ordinary hysterectomy and the radical operation may be readily understood when compared to a simple mastectomy and the modern radical

operation for carcinoma of the breast. Simple hysterectomy for cancer of the cervix is followed by fatal recurrence in nearly 100 per cent of cases.

The safe use of radium, however, requires judgment and experience. A thorough understanding of its action and an appreciation of the complexities of its safe and efficient application is necessary, otherwise we assume a grave responsibility in not giving these sufferers the full benefit of our present day knowledge. If a correct dosage and filtration is used the cancer cells will be destroyed, but *not* the normal tissues. If too great a dosage and insufficient filtration is employed, the normal structures will also be destroyed producing extensive necrosis, septic absorption, hæmorrhages and fistulas, and perhaps death. If too small a dosage is used there will be a failure to destroy all the cancer cells.

Radium is therefore a two-edged weapon and proper training and experience in the treatment of cancer of the uterus by radium therapy is just as essential as in surgery.

Madame Curie has called attention to the great dangers of the improper use of radium, and Regaud has sounded a warning that "it is necessary to have much experience to obtain from this method of treatment all the good that it may give without the evil that it may do."

The difficulties of the safe employment of radium may be likened to Virgil's lines,—

"There on the right her dogs foul Scylla hides,
Charybdis roaring on the left presides."

GEORGE GRAY WARD.

COMPOUND FRACTURES

A COMPOUND fracture nearly always places the surgeon "on the horns of a dilemma." Two problems confront him: first, a broken bone, the treatment of which suggests one fairly well recognized set of procedures; and, second, a wound of the soft parts, usually infected, calling for another generally accepted mode of treatment. Unhappily, these indicated methods of treatment are frequently contradictory, and the problem arises thus: Shall the wound be treated first, and then the fracture; or shall the fracture receive primary consideration? Or can the two be treated simultaneously?

Our teachings here have lacked harmony. To some men treatment of the wound is always primary; the fracture itself can wait. Others, especially orthopedic surgeons, have been unwilling to neglect the fracture and have attempted various compromises in the effort to treat both the fracture and the wound at once. It is apparent, however, that neither the one policy of treating the wound first and then the fracture, nor the other of endeavoring to treat both at once—that is to say, if the wound is treated by the usual antiseptic methods now in vogue—has given us the results that should be obtained in such cases. Recent developments in the management of compound fractures and other infected wounds suggest, however, that combined treatment of fracture and infected wound is possible, and that such a plan of treatment conforms to sound surgical principles. We shall return to a discussion of this plan later.

First, let us summarize the fundamental principles of therapy in compound fractures. In the treatment of all fractures, *as fractures*, the attempt is made, first, to effect an immediate restoration to correct length and position of the fractured part; second, to maintain that correct length and position during the period of healing; and, third, to afford complete rest to the injured part, both to preclude muscle spasm and pain and to facilitate the healing process by avoiding disturbance of the forces

of repair. To make these principles effective we employ various devices which will insure rest, immobilization, and maintenance of correct position with an eye to ultimate restoration of function.

Our treatment of the wound, apart from the fracture, has called for compliance with other principles of therapy. These include, first, primary asepsis or antisepsis to exclude or reduce infection; second, drainage; and third, wound treatment to prevent, reduce or control infection of the wound. But here arises the dilemma! The usual methods of treating the *wound* conflict with what we know should be the correct treatment of the *fracture*, and in the methods now generally employed there is no solution of this conflict.

Some surgeons, as we have said, prefer to treat the wound first and the fracture secondarily. This means delay in reducing the fracture, disturbance, for antiseptic dressings or treatment, of whatever fixation or immobilizing apparatus is used, irritative movement of the injured and inflamed part, frequent exposure of the wound surface to injury and infection, and, too often, failure to obtain union of the fracture, or union in malposition.

Of those methods, hitherto employed, which attempted a compromise and endeavored to deal with fracture and wound at the same time, we can say only that it has been our observation that they have sacrificed the best principles of treatment for one or the other, and have led to results as poor as those described above. To afford access to the wound, control in splints has been sacrificed and there has been no true immobilization. There has thus been no efficient maintenance of fracture fragments, no real protection against muscle spasm, and no provision for rest for the patient. Moreover the wound itself—and here is the chief reason for failure—has been treated too much. We have failed to appreciate that the wound surface also requires protection and rest, and that frequent flushings and dressings do more harm than good. Those compromise methods of treatment, therefore, which call for complicated splinting devices arranged to permit daily or more frequent dressing of the wound, or plaster-of-Paris casts with large windows to allow for the insertion of Carrel-Dakin tube dressings and irrigations, fail to observe principles of fracture therapy which are fundamental: rest, immobilization, protection against new infection, and the others which have been listed.

Our own experience with the treatment of osteomyelitis in all its forms has convinced us that both the fracture and the wound in infected compound fractures can be treated with full observance of the correct surgical principles which we have outlined. We have demonstrated that frequent dressings, irrigations, and other disturbances of the wound are unnecessary; indeed, they violate these principles. By dispensing with such procedures we are able to control both the fracture fragments and the patient, and to facilitate healing of the wound. We use plaster-of-Paris casts combined with our method of "closed drainage"; the wound is packed open with sterile vaseline gauze, while the fracture is controlled when necessary by ice tongs, pins, or moleskin traction straps which are included in the plaster cast. Nor do these devices have to be disturbed at any time during the course of treatment. These are really effective as traction and immobilizing devices.

It may be admitted that skeletal devices used in conjunction with Thomas splints and Balkan frames have, in the hands of fracture specialists such as Sinclair and Pearson, and in such special fracture clinics as the one at the Massachusetts General Hospital, given satisfactory results. But this has depended largely upon the exceptional skill in their

original application and the careful supervision throughout treatment made possible by the kind of after care we developed in a few of the military hospitals in 1918–1919. With most surgeons, however, due to a lack of similar skill in applying the original apparatus, to difficulty of controlling the patient during treatment, and to all of those factors which tend to separate the patient from his original attending surgeon, this sort of treatment leads to poor results.

Some surgeons, too, have been able to show good functional results in patients whose fracture fragments were permitted to heal in malposition, but it can hardly be argued that this is a safe course to follow routinely. Several writers have urged less-than-perfect reduction, particularly in children, on the theory that compensatory adjustment of both length and position will occur in the course of growth. Even in adults occasional good results may be had. Extensive studies indicate, however, that healing in correct anatomical position must be obtained if a high percentage of good functional results is to be expected. And in view of the widespread use of the X-ray in checking results and the growing frequency of suits for malpractice, it is becoming increasingly evident that we must make a better showing — anatomically and functionally — than we have in the past.

We believe, then, that we are now in a position to fulfill simultaneously the surgical requirements for sound treatment of compound fractures and their associated infected wounds. We believe that there should be a more extensive use of these fundamentals of surgery: first, adequate primary reduction of the fracture itself; second, primary cleansing of the wound by débridement; third, provision for "closed drainage," that is, efficient drainage under a well-fitting plaster cast; fourth, fixation by means of efficient skeletal devices included and immobilized in plaster of Paris; and fifth, discontinuance of programs of frequent antiseptic flushings or dressings which disturb the healing wound and, all too often, introduce new and secondary infection. It has been shown that such a plan of treatment can be well standardized and used routinely. If this is done the results of treatment will afford us all, both patients and surgeons, considerably more satisfaction than they have in the past.

H. WINNETT ORR.

EARLY AMERICAN MEDICAL SCHOOLS

THE DEVELOPMENT OF THE HARVARD MEDICAL SCHOOL

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FROM a perspective of three centuries it sometimes seems strange that so many years should have elapsed between the founding of Harvard College in 1636 and the beginnings of medical education there in 1782. It should be remembered, however, in the first place, that at the time the New England colonies were settled, the science of medicine was in chaos the world over. The soundest medical training which was to be had was an apprenticeship with an older physician of intelligence and experience. Didactic teaching was merely supplementary to practical but fragmentary experience. Such a situation persisted until within the memory of men now living.

In the second place, the poverty of most of the early settlers and the sparse and scattered nature of the population almost precluded sole dependence on medicine as a livelihood. Usually the minister had a smattering of medicine, either picked up by chance observation or as a result of the brief medical studies often included in the courses of theology.[1] Until well into the eighteenth century, the majority of non-clerical physicians were farmers in the intervals of their professional duties. Further, the University shared in the general stringency and its funds barely sufficed for the support of its three professors.[2]

With the great awakening of interest in science which took place in the middle of the eighteenth century, it was but natural that some thought should be given to the establishment of medical teaching at Harvard. Gifts of medical books, anatomical specimens and the like began to be recorded and it seems obvious, though nowhere expressly stated, that the officers of the College had in mind instruction in some branches of the study of medicine. In 1770 Ezekiel Hersey, a physician of Hingham, left one thousand pounds to the College for the express purpose of aiding in the support of a professor of anatomy and physic.[3] About this time also, dissections began to be carried on in a rather clandestine way by groups of undergraduates. The actual founding of a chair of anatomy was delayed, however, perhaps for lack of a suitable incumbent, but doubtless also on account of the unsettled political situation, until 1782. The lamentable condition of the military medical services during the Revolution was fresh in everyone's mind; medical schools had been founded in Philadelphia and in New York; New England was entering upon a period of prosperity; and John Warren had appeared upon the scene.

John Warren, the first of four generations of the same name[4] to serve the Harvard Medical School, was the younger brother and pupil of Dr. Joseph Warren, who refused the title of surgeon general of the Massachusetts forces to die in the ranks at Bunker Hill. John Warren himself rendered invaluable services as superintending surgeon to the military hospital in Boston, at the age of 24 years. In the army, he had met Morgan, the founder of the University of Pennsylvania Medical School, and was familiar with Morgan's inspiring book on the needs and methods of medical education. Although his formal education in medicine was scanty, he had a profound knowledge of anatomy, in addition to rare ability as a surgeon and teacher. With these gifts he combined the imagination to plan an institution for the future needs of the community, and the courage and persistence to fight for it against all obstacles. Willard, the president of the College, turned to him for advice in formulating a program for the new courses in medicine, and there was little surprise (though much envy) among his colleagues when he received the appointment of professor of anatomy and surgery on November 22, 1782.

[1] Thus Cotton Mather, a clergyman, was largely responsible for the introduction of small pox inoculation in Boston by Dr. Zabdiel Boylston at almost the same time that Lady Wortley Montague initiated the practice in England.

[2] Occupying the chairs of divinity, mathematics, and oriental languages, respectively.

[3] This bequest dwindled during the Revolution and the Hersey professorship of anatomy and physic was endowed in 1792 by his widow and his brother.

[4] J. Mason Warren, the third in line, though an outstanding surgeon, had no connection with the Harvard Medical School. Thomas Dwight, Parkman professor of anatomy from 1883 to 1910, was also a direct descendant of John Warren.

Fig. 1. John Warren—Hersey Professor of Anatomy and Surgery, 1782–1815.

Fig. 2. John C. Warren—Hersey Professor of Anatomy and Surgery, 1815–1847.

Fig. 3. John Collins Warren—Moseley Professor of Surgery, 1899–1907.

The new professor's duties proved arduous. During the 4 months' course of lectures, he had to leave a busy practice and drive from Boston to Cambridge—then a long journey. He did his own dissecting, delivered a lecture often lasting 3 hours, and returned. In addition, he had to provide the cadavers, some of which, it is to be feared, were come by in devious ways.

Two other professors were appointed a little later. The professor of theory and practice of physic was Dr. Benjamin Waterhouse. He was also a young man, the nephew of Fothergill of London, and largely trained abroad. A rather confused picture of his personality has been handed down to us. There can be little doubt but that he was pompous and quarrelsome, but this should not make us forget his scholarly attainments, which were ahead of his day. He championed the cause of vaccination, for example, and won local recognition of its virtues at a time when the remainder of the world was incredulous—the first important scientific achievement of which the new school could boast.

The third member of the medical faculty was Dr. Aaron Dexter, professor of chemistry and materia medica—a distinguished physician and able teacher.

A student who attended—or at least bought tickets for—the 4 months' course of lectures and demonstrations given by these three gentlemen, in addition to the regular college course, received the degree of Bachelor of Medicine. To acquire a doctor's degree he had to go abroad to study.[1] Meager as this training seems, when judged by modern standards, it was not very different from the type of instruction offered in the Old World. There was, for example, only one medical college in England and the course consisted of 72 lectures.

Fig. 4. John Warren—Associate Professor of Anatomy, 1910–1928.

A new standard of medical education was formulated in the early years of the nineteenth century by two young men, just returning from studies abroad: Dr. John Warren's son, John Collins Warren, and James Jackson. They were of about the same age and had come to form a close friendship while studying at St. Thomas' in London under William and Astley Cooper—a friendship which lasted throughout a long lifetime. Warren resembled his father: he was an energetic, determined reformer, a teetotaler, and a scientist of rigid standards. Jackson, "the beloved physician," had a more serene, warmer, and more sympathetic temperament. The difference is reflected in their published works. Warren's masterpiece is his *Surgical Observations of Tumours* (1837), perhaps the ablest treatise on surgical

[1] A few honorary degrees were awarded, chiefly to established practitioners.

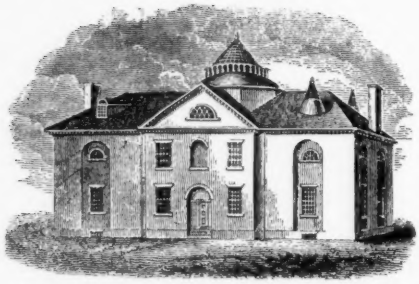

Fig. 5. The Massachusetts Medical College, 1815.

pathology of the pre-microscopic era. Jackson's *Letters to a Young Physician* (1855) are a series of essays on the philosophy and art of clinical medicine, quite unsystematic but charming and full of hints of value to every physician now as then. Warren among other achievements recorded the first successful operation for goiter in this country, founded the School's pathological museum and performed the first operation under ether in public. Jackson recommended the open air treatment of tuberculosis and described alcoholic neuritis. Both had high ideals of medical practice and education, and their talents were mutually complementary.

Largely through the efforts of the Warrens, Jackson, and a few other open minded physicians, the necessity of further provision for clinical instruction was impressed on the Corporation of the University. At this time, the only institution in the vicinity available for clinical instruction was the almshouse in Boston. On this account and because of the greater number of physicians in the larger city, it seemed expedient to move the medical institution there. The change was made in 1810. The younger Warren had already been appointed his father's assistant, and now Jackson was made professor of clinical medicine. The lectures were given in rented rooms "over the store of White, the druggist, at 49 Marlborough Street."[1] Two years later Waterhouse resigned, no longer on speaking terms with the rest of the faculty, as the result of an unfortunate controversy, and Jackson was appointed to the chair of theory and practice of physic in his place.

The city almshouse was lamentably deficient for teaching purposes, and the temporary rooms in which medical lectures were held were thoroughly unsatisfactory. In 1810, Jackson and Warren circulated an appeal for funds to build a suitable General Hospital, but donations came slowly. Petitions to the State legislature and to the Corporation of the College for a more suitable building for didactic instruction were granted in 1814, and the following year a small, but for the period, well equipped school was completed on Mason Street, near the Common. As the State had contributed so largely to its expenses, its name was changed from "The Medical Institution of Harvard University" to "The Massachusetts Medical College." Harvard contributed a small sum to its maintenance and had a correspondingly small control over its affairs. For the most part it was a private venture—financially unprofitable—on the part of the Faculty, which undertook to meet the upkeep of the building and materials for instruction out of fees received directly from students.

The death of John Warren, the founder of the school, occurred in the same year.

In 1821, the Massachusetts General Hospital was finally built. The story of this institution, which has played so large a rôle in the development of medicine in America, will be told in a later article in this series. The facilities for clinical instruction now became equal to any in the country and pupils were attracted from all parts of the United States.

An important accession to the faculty of this period was Jacob Bigelow, professor of materia medica. He was a man of broad culture and many interests, a botanist as well as a physician, the founder of the great Mt. Auburn cemetery (the first of its kind in the country) and the father of Henry Jacob Bigelow. His most noteworthy medical contribution was his paper on *Self-Limited Diseases*, which went far toward bringing an end to the days of "a pill for every ill."

Even with the new equipment and an unusually able group of lecturers, the lack of a longer course and of individual study made itself felt. To meet this need, a number of private medical schools arose during the first half of the century. While they varied greatly in quality, some of the better ones grew to be recognized as contributing greatly to medical education. The most famous one, the Tremont Street Medical School, which included in its faculty such renowned teachers as Oliver Wendell Holmes, Henry J. Bigelow, Jeffries Wyman, and Charles and J. B. S. Jackson, finally incorporated into Harvard as the Summer School.

Aside from inevitable changes in the faculty which cannot be reported here, the school continued without fundamental change until 1846. In that year, three important events occurred.

[1] Now Washington Street.

Fig. 6. Oliver Wendell Holmes, Parkman Professor of Anatomy and Physiology, 1847–1882.

Fig. 7. Henry J. Bigelow, Professor of Surgery, 1829–1882.

The first, which marked the beginning of a new era in medicine, was the demonstration of the anæsthetic properties of ether. The sordid personal details of this magnificent discovery are familiar to all and need not concern us further. The two others were of local significance only but represented a turning point in the history of the school. One was the transfer to a new building, and the other was the accession of a group of brilliant young men to the faculty.

It had been obvious for some time to everyone concerned that the little building on Mason Street, in spite of enlargements, was too small for its purpose. Money was accordingly advanced by the University to build a new one, and was to be repaid by the proceeds of the sale of the old. The balance still owing was to be paid off gradually by the faculty of the medical school. Accordingly, a much more commodious building was erected on the site of the present out-patient department of the Massachusetts General Hospital, on land presented to the school by Dr. George Parkman, a wealthy practitioner. Three years later it was the scene of his murder by Dr. John Webster, Erving professor of chemistry. But that is a story aside from our present narrative.

About the time that the School moved to its new quarters, three new professorships were created, one combining anatomy and physiology, one of anatomy alone, and one of pathology—the first in the country. The significance of this change was not alone that further instruction was given in the fundamental, preclinical sciences. A greater innovation was that the new professors did not merely teach in the intervals of private practice, but devoted all their working hours to study and instruction—the beginning of the full time system. The choice of men to be thus honored was particularly happy. Oliver Wendell Holmes was chosen for the chair of anatomy and physiology, Jeffries Wyman for that of anatomy, and John B. S. Jackson for that of pathology.

No more than a hint can be given here of Holmes' many sided genius. He was probably the wittiest lecturer that has ever adorned the University. Anatomy is still enlivened by some of his apt metaphors and droll comparisons. He was beloved by his students and in turn considered no labor too great which would simplify his subject or make it more interesting. He had talent for original observation and invention, as is shown by his essay "On the Contagiousness of Puerperal Fever" (1843), by his demonstration of the cells in bone (1847) and by his ingenious stethoscope and student's microscope. But he preferred teaching. In addition to anatomy and physiology, he lectured on microscopy and psychology. In 1857, he founded *The Atlantic Monthly* with James Russell Lowell and from that time on became increasingly engaged in literary pursuits.

Jeffries Wyman was also a good teacher, but his chief interest was in his anatomical laboratory. He was an incessant worker and published no less than 175 articles on comparative anatomy. He was the first to describe and name the gorilla (1845), among other achievements.

John Barnard Swett Jackson was also a devoted scientist. His publications are few, but he has left a monument in his arrangement of the Warren Anatomical Museum which was donated to the school in 1847. His catalogue of the Museum, published in 1870, is in reality a textbook of pathology.

In 1849, Henry Jacob Bigelow was appointed to the chair of surgery. He was the first to publish an account of Warren's operation under Morton's ether and made many contributions to the subject of anæsthesia. His best known innovations in surgery are his method of reducing dislocations of the hip by taxis and his instruments for litholapaxy. If his lectures lacked the sympathy and wit of Holmes' or the scientific depth of Wyman's, they made up for it in brilliance and wealth of demonstration. His clinical judgment and operative dexterity were unsurpassed. Tall, handsome, well dressed, and driving a French chaise with horses in tandem, he was known by sight to every inhabitant of the city. He exercised a sort of benevolent tyranny over the school, hospital, and practice of surgery throughout New England.

There was a gradual expansion and improvement in the School for the next two decades and the addition of such able practitioners to the faculty as George Cheyne Shattuck, Henry I. Bowditch, John Ware, Humphreys Storer, and Calvin Ellis. The Boston City Hospital was opened in 1864 and added its profusion of clinical opportunities to the resources of the School.

The basis of operation of the School remained essentially what it was under Warren and Jackson: a private institution supported by students' fees with scarcely more than a nominal connection with the University. Original investigation was a by-product. The aim of the School was to supplement the instruction in practical medicine that could be acquired by assisting an older physician. The course of lectures was the same each year. To be examined for a degree in medicine, a candidate had to show that he had bought tickets for two such courses of lectures and had spent 3 years with a practicing physician. The examinations were given by the teachers who gave the courses and received the fees and the candidate needed to pass in only a majority of subjects to receive his degree. This was the case not only at Harvard—where fairly high standards were maintained—but throughout the country and in many schools with miserable equipment and commercial ideals. The Chicago Medical College in 1859 introduced a 3 year graded course with required hospital attendance, but this was a solitary example.

A protest was made in 1866 by James C. White, later professor of dermatology. He pointed out the low general standard of medical practice and the fact that the only really qualified teachers of medicine were those who had gone abroad to study "where medicine seemed a strange, almost a new science to them, and where years might be spent in learning what was then taught in but a single subdivision of one of the old general departments."

The real cataclysm came in 1870 following the accession of Charles W. Eliot to the presidency of the University. He had a first hand knowledge of the methods of the medical school, as he had acted as lecturer in chemistry there in 1856. No description of the transformation could equal that which Holmes has left us:

> Our new president, Eliot, has turned the whole University over like a flapjack. There never was such a *bouleversement* as that in our Medical Faculty. The Corporation has taken the whole management of it out of our hands, and changed everything. We are paid salaries, which I rather like, though I doubt if we gain in pocket by it. We have, partly in consequence of outside pressure, remodeled our whole system of instruction. Consequently, we have a smaller class, but better students, each of whom pays more than under the old plan of management. It is so curious to see a young man like Eliot, with an organizing brain, a firm will, a grave, calm, dignified presence, taking the ribbons of our classical coach-and-six, feeling the horses' mouths, putting a check on this one's capers, and touching that one with the lash—turning up everywhere, in every Faculty (I belong to three) on every public occasion, at every dinner *orné*, and taking it all as naturally as if he had been born President. . . .

Reform was bitterly and doggedly opposed by Bigelow and by those of the Faculty that owed their positions to him. In an able defense of the established position, Bigelow said on one occasion:

> Whatever else it may do or not do, a medical school should aim first, then to give a plain, sound, solid education, without error, if without ornament. For, in the first place, you cannot do better than this. It is the highest average development of which the mass of the material you are dealing with is susceptible, in view of the character of its preliminary education and of the accepted three years' term of study. In the next place, you need not do better . . . . Without good judgment, for which education is not a substitute, if you fill the mind of the student with Chemistry and Physiology and Drugs, as leading ideas, the chances are that he will apply this collateral, imperfectly applicable knowledge wrongly and that he will have to forget and abandon much of it before he gets down to a working medical level. . . . Medical discovery is generally not made by workers in chemical and physiological fields, but by subsequent and more purely medical workers, who apply to disease the materials and results of such previous work.

The school would be the loser if the simple, practical point of view here expressed were ever

Fig. 8. The Harvard Medical School.

lost sight of. Fortunately, it has not proved incompatible with the results of reform.

Eliot had his way. The president of the University became an active member of the faculty of medicine, and all appointments had to pass the Overseers and Corporation of the University. The school's funds were received and disbursed by the university, though still under direction of the faculty. The course was lengthened to 3 years, the length of the usual college course, and the instruction was graded for each class. Each student was required to perform a certain amount of laboratory and clinical work. Examinations were required to pass from one class to another and for graduation. The school was thus placed on a full university basis and the modern era of medical education was begun.

The expansion of the faculty necessitated by the new order, and increased year by year up to the present, makes it impossible even to mention all the names of the procession of eminent practitioners, able scientists, and revered teachers, who maintained the standards set by the founders of the school. I can give only an outline of the physical changes and developments in policy of the school and the more outstanding scientific discoveries of the last 60 years.

The expansion of the curriculum brought about by the new order led to the creation of professorships of a number of specialties, at that time scarcely recognized in this country. Chairs of dermatology, mental diseases, and ophthalmology were established in 1871, occupied by White, Tyler and Williams, respectively. A particularly important development was the inauguration of a physiological laboratory for teaching purposes under Henry P. Bowditch in 1871—apparently the first one in America. Bowditch was made a full professor in 1876, and retained this title until 1906.

Instruction was begun in other special subjects, such as pediatrics, laryngology, and otology, though on a smalier scale. As the school was now intended to replace teaching by apprenticeship, the amount of individual instruction and section work underwent a progressive increase which still continues. Meanwhile, the tradition of didactic teaching by lectures and demonstrations was ably carried on by Francis Minot in medicine and David Cheever in surgery.

The new order made the Grove Street building immediately out of date. Fortunately, a large fund of gifts and bequests was available for the construction of a new one. Construction was begun in 1881 at the corner of Boylston and Exeter Streets, next to the site of the present Public Library. The total project cost over $300,000. The building was dedicated in 1883, the centenary of John Warren's first lectures.

A fourth year of instruction was made optional in 1881 and compulsory in 1891. The requirements for premedical education were stiffened and an examination was given before matriculation. About this time, the Lying-in Hospital, the Children's Hospital, and the Free Hospital for Women, in succession permitted instruction to students within their walls. The chairs of gynecology under Baker, of laryngology under

Knight, and of otology under Blake were all established in 1888. Bacteriology was recognized for the first time in this country by the appointment of Harold Ernst as demonstrator in 1885 and professor in 1895. Under his direction, J. Collins Warren prepared and used sterilized dressings in 1887. Two particularly famous departments were established in 1893: those of pediatrics under Rotch and of orthopedics under Bradford. The same year, a department of diseases of the nervous system under James Putnam offered instruction in neurology as well as in psychiatry. Among the outstanding teachers of this period were Thomas Dwight, in gross anatomy; Charles S. Minot, in embryology; Reginald Fitz, in medicine, and Maurice Richardson, in surgery.

The requirements for admission to the medical school were increased in 1901 to include the degree of A.B. or its equivalent. The step did not prove an unqualified success. It was felt by many members of the faculty that some students who for one reason or another were not able to spend 4 years at college were in fact more desirable candidates than others who had complied with the new standards. Accordingly, in 1916 the requirements were altered to the extent that a man who had done the equivalent of 3 years' college work and stood in the first third of his class would also be eligible for admission. This amount of flexibility has proved highly satisfactory.

The fruits of expansion were seen in investigative work as well as in instruction. The pathological and clinical picture of acute appendicitis was firmly established about this time by Fitz. Bowditch demonstrated the non-fatigability of nerve in 1892; Cannon took the first X-rays of the digestive tract in 1897, and Theobald Smith differentiated between human and bovine tubercle bacilli in 1898. During this period Rotch was developing his important percentage system of infant feeding.

As the end of the century approached, it became obvious that the Boylston Street building was already growing too small. Funds were therefore again solicited for a more extensive plant. Something over $4,000,000 was collected, Morgan and Rockefeller being the largest donors. A tract was purchased just beyond the Fenway, at that time surrounded by most repellent dump heaps. Enough land was acquired to accommodate the Dental School, Children's Hospital, and a university hospital at a later date. The funds were raised and plans were made largely by J. C. Warren and H. P. Bowditch.

A description of the beautiful group of five buildings which was completed in 1906 would be superfluous. They still afford adequate space after a quarter of a century, and plans are ready to double their capacity if the necessity should arise.

The need for a hospital intimately connected with the medical school had long been felt. A bequest from Peter Bent Brigham became available for this purpose shortly after the opening of the new buildings. The hospital, called by his name, was built adjacent to the school and opened in 1913. About the same time, the Children's and Infants' Hospitals were moved to new buildings near the school. Just across the street, a new Lying-in Hospital was built in 1924 and a most comfortable dormitory for students in 1928. The sumptuous Beth Israel Hospital was opened the following year only a minute's walk away. The opportunities for clinical instruction now offered at Harvard are equalled by few schools, in this country or abroad.

Of recent scientific achievements, perhaps the most outstanding have been the contributions of Dr. Harvey Cushing to the surgery of the nervous system and physiology of the hypophysis; of Drs. George R. Minot and William P. Murphy on the treatment of pernicious anæmia with liver; of Dr. S. Burt Wolbach on the etiology of Rocky Mountain spotted and typhus fever; and of Dr. Hans Zinsser on the etiology of typhus. The buffer system of the blood has been worked out by Dr. Lawrence J. Henderson, and Dr. Walter B. Cannon has filled out the gaps in the physiology of the sympathetic nervous system. The funds available for investigation have steadily increased and the extent of research projects now in progress is far beyond the scope of this paper.

The history of the Harvard Medical School has been one of constant growth—not so much in the number of students, for there were classes larger than the present ones in the Boylston Street days, but in resources, instructors, and investigators. The school already offers far more facilities for instruction than any one student can use. If growth continues, what will the school of the future be like? Will it become more and more an institution for research until medical education dwindles to a by-product? That would be a loss to research. Will it undertake to offer instruction to a larger number of students, by continuing formal instruction through the summer as has been several times suggested? Perhaps, but the proportionate increase in funds limited to research may continue nevertheless. Will it lay more emphasis on graduate study, not necessarily in classes, but by extending the facilities of laboratories and clinics to increasing

numbers of qualified graduates who desire further special opportunity and individual instruction? We may be sure at all events that the School will not stand still.

NOTE.—Most of the material for this sketch has been drawn from Harrington's compendious history (*The Harvard Medical School; a History, Narrative and Documentary.* New York: Lewis Publishing Co., 1905. 3 vols.) to which I gratefully acknowledge indebtedness. I have also drawn on biographies and shorter articles too numerous to mention in detail. Dr. David Cheever has been good enough to revise the manuscript and add many suggestions.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

THE tenth *Scientific Report of the Imperial Cancer Research Fund*[1] presents the results of investigations conducted during the past 3 years by various members of the laboratory staff.

Ludford found the lowering of resistance to the growth of transplantable tumors to be the result of some interference with the function of cells derived from lymphocytes and monocytes which segregate acid dyes. These cells are seen in large numbers around the margins of absorbing tumors and are particularly common in tumors that have been exposed to radiation. These observations indicate the possible danger in the treatment of cancer with colloids under circumstances in which the body may be offering some resistance to the malignant process.

Ludford also studied the differential reaction of normal and malignant cells *in vitro* to Trypon blue and observed that malignant cells do not segregate this dye in the same manner as non-malignant cells when both are subjected to the action of the dye under identical conditions in tissue culture. The cells of filterable tumors fail to take the vital stain *in vivo* or *in vitro* whereas their non-malignant prototypes segregate the dye. Ludford suggests that the failure of malignant cells to segregate acid dyes may be due either to their impermeability to these substances or possibly to some peculiarity of metabolism possessed by the cancer cell.

Fould studied the effects of vital staining upon the distribution of the Brown-Pearce rabbit tumor and discovered that tumors are rarely found in the spleen and are relatively uncommon in lungs and liver. Vital staining with Trypan blue greatly increased the incidence of tumors in all these organs after intravenous inoculation. The author suggests that the spleen in man resists the establishment of secondary deposits by a local mechanism. There is considerable evidence to support the view that the cells of the reticulo-endothelial system play a predominant rôle in this mechanism.

Cramer has studied the mode of action of radium upon precancerous lesions and finds that radium delays and sometimes inhibits the development of cancer in an area of skin which is the seat of a precancerous state. Investigations were made in an effort to determine whether the application of radium breaks down the resistance of normal skin to the development of cancer. No evidence could be adduced to support this view.

Cramer also conducted further studies upon the therapeutic action of radium and concludes that regression of tumors following irradiation occurs as the result of a combined effect upon the tumor cells and upon the tumor bed. These observations are supported by Ludford's cytological studies from which it appears that in addition to a direct action of radiation upon tumor cells, there is an important effect of radiation upon the stroma and particularly upon the blood vessels.

Interesting experiments are reported by Crabtree upon respiration and carbohydrate metabolism of irradiated tumor cells *in vivo* and *in vitro*. The author finds that radium causes a lowering of the respiratory function of cells and to a certain extent of their glycolytic function also. Radium irradiation exerted no differential effect upon metabolism of tumor tissue as against normal tissue. The conception that tumor cells are inherently more vulnerable than normal tissue fails to be supported by these studies. (Clinical and pathological evidence strongly support the view that certain tumor cells which we term radiosensitive are inherently more vulnerable to radiation than are normal cells.)

MAX CUTLER.

THE sixth edition of Zachary Cope's *Early Diagnosis of the Acute Abdomen*[2] bears evidence of both the author's desire to keep the text up to date and of the welcome reception that the book has received. The fact that no considerable alteration has been necessary is indicative of the firm ground upon which the work is based. As is so typical of the English school of surgeons, the author's diagnoses are based, in so far as possible, upon our knowledge of anatomy and physiology of the abdomen and its contents, with somewhat greater emphasis placed upon anatomy than upon physiology. The book is not encumbered with numerous complicated tests or descriptions of questionable and confusing diagnostic procedures, but contains a clear, practical, and concise discussion of the disease conditions which comprise the acute abdomen, or which may be confused therewith. The volume will long remain a classic monograph.

MICHAEL L. MASON.

[1] TENTH SCIENTIFIC REPORT ON THE INVESTIGATIONS OF THE IMPERIAL CANCER RESEARCH FUND. Under the direction of the Royal College of Physicians of London and the Royal College of Surgeons of England. London: Taylor and Francis, 1932.

[2] OXFORD MEDICAL PUBLICATIONS. THE EARLY DIAGNOSIS OF THE ACUTE ABDOMEN. By Zachary Cope, B.A., M.D., M.S.(Lond.), F.R.C.S. (Eng.). 6th ed. New York and London: Oxford University Press, 1932.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

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Franklin H. Martin, Chicago, *Director-General*
Philip H. Kreuscher, *Chairman;* Oscar E. Nadeau, *Secretary, Committee on Arrangements*

PROGRAM FOR THE 1933 CLINICAL CONGRESS IN CHICAGO

CLINICAL CONGRESS PROGRAM IN BRIEF

Sunday, October 8

7:00. Health meeting, A Century of Progress.

Monday, October 9

10:00. Hospital conference.
2:00. Clinics in hospitals.
2:00. Hospital conference.
2:00. Surgical film exhibition.
4:30. Fracture demonstration, illustrated.
8:15. Presidential meeting.

Tuesday, October 10

9:00. Clinics in hospitals.
9:30. Hospital conference.
10:00. Surgical film exhibition.
11:30. Fracture demonstration, illustrated.
2:00. Clinics in hospitals.
2:00. Demonstration in administration at hospitals.
2:00. Surgical film exhibition.
2:30. Symposium on treatment of fractures.
8:15. Scientific session, general surgery.
8:15. Scientific session, section on otolaryngology.

Wednesday, October 11

9:00. Clinics in hospitals.
9:30. Hospital conference.
10:00. Surgical film exhibition.
11:30. Fracture demonstration, illustrated.
11:30. State and provincial executive committees.
2:00. Clinics in hospitals.
2:00. Demonstration in administration at hospitals.
2:00. Surgical film exhibition.
2:30. Symposium on curability of cancer.
4:30. Fracture demonstration, illustrated.
8:00. Community health meeting—Chicago Stadium.
8:15. Scientific session, general surgery.

Thursday, October 12

9:00. Clinics in hospitals.
9:30. Hospital conference.
10:00. Surgical film exhibition.
11:30. Fracture demonstration, illustrated.
2:00. Annual meeting of College.
2:00. Clinics in hospitals.
2:00. Demonstration in administration at hospitals.
4:00. Reception at American College of Surgeons.
4:30. Fracture demonstration, illustrated.
8:15. Scientific session, general surgery.
8:15. Scientific session, section on ophthalmology.

Friday, October 13

9:00. Clinics in hospitals.
10:00. Surgical film exhibition.
10:00. Meeting of new Fellows, class of 1933.
11:00. Symposium on urological surgery.
11:30. Fracture demonstration, illustrated.
2:00. Conference on industrial medicine and traumatic surgery.
2:00. Clinics in hospitals.
2:00. Surgical film exhibition.
4:30. Fracture demonstration, illustrated.
8:15. Convocation.

THE surgeons of Chicago will present for the twenty-third annual Clinical Congress of the American College of Surgeons, October 9–13, a program of clinics and demonstrations in the hospitals and medical schools that will provide a complete showing of the clinical activities in all departments of surgery in this great medical center. They are keenly interested in outdoing all previous efforts, and in making its plans the Committee on Arrangements has the hearty cooperation of the clinicians in the medical schools and more than fifty hospitals that will participate in the clinical program.

A schedule of operative clinics and demonstrations as prepared by the Committee is presented in the following pages. It will be noted that clinics are scheduled to began at 2 o'clock on the afternoon of October 9 continuing through the four following days with sessions both morning and afternoon.

The clinical program contains many features of special interest including: (1) Cancer clinics demonstrating the treatment of cancer cases by surgery, radium and X-ray; (2) fracture clinics where modern methods in the treatment of fractures will be demonstrated; (3) clinics in trau-

matic surgery demonstrating the newer methods of rehabilitation by surgery and physiotherapy of patients injured in industrial, automobile and other accidents.

The clinical program as published at this time is merely an outline or basis for the final program, as during the Congress the clinical program will be published daily in the form of bulletins prominently displayed on large bulletin boards at headquarters at the Stevens Hotel. These bulletins will be posted each afternoon showing in complete detail the clinics to be given on the following day. The same material will be published in the *Daily Bulletin* for distribution to the visiting surgeons early each morning.

Special features of the general program for the Congress include: (1) A conference on fractures on Tuesday afternoon arranged by the College Committee on the Treatment of Fractures; (2) a symposium on the curability of cancer on Wednesday afternoon; (3) a symposium on urological surgery on Friday morning; (4) a symposium under the auspices of the Board on Industrial Medicine and Traumatic Surgery on Friday afternoon. Complete programs for these conferences and symposia appear in the following pages.

CANCER IS CURABLE SYMPOSIUM

An outstanding feature of this year's Congress —of great interest to all cancer workers and to the public as well—will be a symposium on the curability of cancer to be held in the ballroom of the Stevens Hotel at 2:30 o'clock on Wednesday afternoon. This will be participated in by a group of clinicians who are especially interested in the treatment of this disease. Each speaker will record his five-year cures of cancer. Clinicians who participate will furnish incontrovertible evidence that cancer is curable, emphasizing that if all cases of cancer were treated in the incipient stage the annual cancer death rate of the United States might be reduced one-third or from 150,000 to approximately 100,000. The program will be found on a following page.

EVENING MEETINGS

Programs for a series of seven evening meetings in the ballrooms of the Stevens Hotel will be found in the following pages.

At the presidential meeting on Monday evening, at which the president-elect Dr. William D. Haggard, of Nashville, Tenn., is to be inaugurated, a number of distinguished visiting surgeons from foreign countries are to be presented. Among those who have indicated their intention of being present are: Prof. R. Marshall Allen, Melbourne, Australia; Dr. Lorenz Boehler, Vienna; Prof. Dr. Eugen Kisch, Berlin; Prof. Rudolf Nissen, Berlin; Prof. Vittorio Putti, Bologna, Italy; Prof. Dr. Wolfgang Rosenthal, Leipzig, Germany; Prof. H. Beckwith Whitehouse, Birmingham, England. A feature of this session will be the annual John B. Murphy oration in surgery to be delivered by Dr. Loyal Davis, of Chicago, whose subject will be "The Story of a Master Surgeon."

At the annual Convocation of the College on Friday evening, at which the 1933 class will be received into Fellowship in the College, the Fellowship address will be delivered by Robert Maynard Hutchins, A.M., LL.D., president of the University of Chicago. The presidential address at the same session will be given by Dr. William D. Haggard, of Nashville, Tennessee.

Two sessions of special interest to ophthalmologists and otolaryngologists will be held in the north ballroom of the Stevens Hotel on Tuesday and Thursday evenings, at which meetings men of outstanding experience in these specialties will present and discuss papers.

FRACTURE CONFERENCE AND DEMONSTRATION

Under the auspices of the College Committee on the Treatment of Fractures, Dr. Frederic W. Bancroft, New York, chairman, a conference has been arranged for Tuesday afternoon at 2:30 in the ballroom of the Stevens Hotel. The detailed program therefor appears on a following page.

The treatment of fractures will also be the subject of daily demonstrations in the Exhibition Hall as arranged by the Chicago Regional Fracture Committee. Illustrated talks on fractures by members of this Regional Committee will be given twice daily at hours to be announced in the *Daily Bulletin*.

Dr. W. Edward Gallie, of Toronto, will deliver the annual fracture oration on Wednesday evening, his subject being "The Treatment of Fractures Involving Joints."

CONFERENCE ON INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY

During the past three years the College has conducted investigations and surveys in large areas of the United States to ascertain present medical conditions in industry and to inform employers of adequate methods. Results of these surveys will be presented by investigators in the symposium at 2:30 Friday afternoon in the ballroom of the Stevens Hotel under the auspices of the Board on Industrial Medicine and Traumatic Surgery, of which Dr. Frederic A. Besley is Chairman.

Other papers in the symposium deal with the clinical aspects of injuries occurring in industry, and methods of rehabilitation of the injured, presented by surgeons of wide experience in this field. The complete program appears on one of the following pages.

COMMUNITY HEALTH MEETING

In recognition of its obligation to the public to provide authoritative information on modern surgery, better hospitals, and prevention of disease, a community health meeting will be held on Wednesday evening, October 11, in connection with the Clinical Congress. For this purpose the Chicago Stadium, which will accommodate approximately twenty thousand, has been secured. A program appropriate for such an occasion has been prepared consisting of brief interesting talks on scientific medicine, health, and hospitals by speakers of note. The complete program for this meeting appears on another page.

ANNUAL HOSPITAL CONFERENCE

The program for the sixteenth annual hospital conference arranged by the Hospital Standardization Department of the College, as presented in the following pages, presents a group of interesting papers, round table conferences and practical demonstrations that deal with the important problems related to hospital efficiency.

The conference opens at 10 o'clock on Monday morning in the ballroom of the Stevens Hotel, continuing on Tuesday, Wednesday and Thursday. Papers will deal with the vital problems affecting administrative, professional and the nursing phases of hospital work, with particular emphasis directed toward professional standards and the highly important problem of medical economics.

Sessions will be held in the ballroom of the Stevens Hotel on Tuesday, Wednesday and Thursday mornings, while for the afternoons an important and interesting series of demonstrations in several of the local hospitals dealing with departmental organization, management and function has been arranged. These clinics in hospital administration afford unusual opportunities for the visitors to see how local hospitals handle their daily routine, and, in comparison, to appraise the efficiency of their own methods.

The program of the conference has been carefully planned to give it a broad interest with a careful selection of subjects to be discussed by eminent authorities in the surgical and hospital field. Greatly increased interest on the part of surgeons in both administrative and scientific phases of hospital work has been evident in recent years. The program to be presented this year will be unique in providing a discussion of many subjects of importance to the three major groups of the hospital—medical, nursing and business. An opportunity is also afforded to chiefs of staffs, heads of departments and members of staffs to participate in a program dealing particularly with the care of the patient, and they may expect to benefit from an exchange of ideas with trustees, superintendents and others concerned with hospital administration.

SURGEONS' WEEK AT A CENTURY OF PROGRESS

A Century of Progress has made an admirable and fitting contribution to medicine and surgery through the medical exhibits in the Hall of Science. Since the opening of the exposition many thousands of people have viewed these exhibits with intense interest and have gone home with a more rational viewpoint of scientific medicine.

Another contribution by A Century of Progress will be Surgeons' Week, commencing October 8, which will be opened by a large assembly in the court of the Hall of Science on Sunday evening, when an appropriate and interesting program will be presented following the Arcturus ceremony. Among other interesting features of this program will be addresses by distinguished surgeons from Central and South America, Australia, Great Britain, and the Continent. Throughout the week at A Century of Progress talks and radio broadcasts will be given by Fellows of the College in connection with the daily program. All the Fellows of the College, their families and friends, are invited to attend the Sunday evening assembly in the court of the Hall of Science.

STATE AND PROVINCIAL EXECUTIVE COMMITTEES

A meeting of the State and Provincial Executives Committees with officers of the College has been called for 11:30 o'clock Wednesday morning at the Stevens Hotel. This meeting is called for the purpose of obtaining information on which may be based the itinerary of the College for its sectional meetings and the desirable grouping of the states and provinces.

EDUCATIONAL AND SCIENTIFIC EXHIBITS

Departmental activities of the College will be demonstrated by means of a series of exhibits located in the Exhibition Hall. These include exhibits by the Committee on the Treatment of Fractures of which Dr. Frederic W. Bancroft, of

New York, is chairman; Committee on the Treatment of Malignant Diseases, Dr. Robert B. Greenough, of Boston, chairman; Registry of Bone Sarcoma, Dr. Dallas B. Phemister, Chicago, chairman; Cancer Clinics, in charge of Dr. Bowman C. Crowell, Associate-Director of the College; Hospital Standardization, in charge of Dr. Malcolm T. MacEachern, Associate-Director of the College; Department of Literary Research.

TECHNICAL EXHIBITION

An extensive technical and educational exhibition under the direction of Mr. A. D. Ballou, General Manager of the Clinical Congress, will occupy the Exhibition Hall on the lower floor of the Stevens Hotel. This exhibition will include surgical instruments and apparatus of all kinds, hospital, laboratory, X-ray and other diagnostic and therapeutic equipment, medical books, pharmaceuticals, etc. A visit to the technical exhibition will provide many suggestions for improving the environment of the surgeon, including the newest in physical, therapeutic and mechanical innovations.

SURGICAL FILM EXHIBITION

Throughout the week surgical motion picture films, both sound and silent, will be exhibited daily at the Stevens Hotel. This showing of films demonstrating clinical features of interest has met with popular acceptance in previous years. Many new films are to be shown. Detailed programs will appear in the *Daily Bulletin*.

MEETING OF NEW FELLOWS

Candidates for Fellowship in the American College of Surgeons, class of 1933, will assemble in the grand ballroom of the Stevens Hotel at 10 A.M. on Friday for the necessary instructions previous to receiving their Fellowships.

RECEPTION AT THE COLLEGE

A reception and tea for Fellows of the College, their ladies and guests of the Clinical Congress will be given on Thursday afternoon from 4 to 6 o'clock at the American College of Surgeons, 40 East Erie Street.

REDUCED RAILWAY FARES

In the states east and southeast of Chicago and in the eastern provinces of Canada, the railways have authorized reduced fares on the certificate plan on account of the Clinical Congress so that the total fare for the round trip will be one and one-third the ordinary one-way fare. To take advantage of these reduced rates it is necessary to pay the full one-way fare to Chicago procuring from the ticket agent when purchasing ticket a convention certificate which certificate is to be presented at headquarters for the signature of the General Manager of the Clinical Congress and a visé of a special representative of the railways. Upon presentation of a viséd certificate to the ticket agent in Chicago not later than October 17, a ticket for the return journey by the same route as traveled to Chicago may be purchased at one-third the one way fare. Tickets may be purchased between October 5 and 11 in the territory covered by this arrangement. The return journey must be completed within thirty days from date of sale of going ticket. It is important to note that the return trip must be made by the same route as used in traveling to Chicago, and that the certificate must be viséd at headquarters during the meeting and return ticket purchased not later than October 17.

Reduction of fares does not apply to Pullman fares or extra fares charged for passage on certain trains. Ticket agents will supply detailed information with regard to dates of sale, rates, routes, etc.

In the western and southwestern states, including the Pacific coast states and the western provinces of Canada, the railways have authorized the sale of round-trip tickets to Chicago at very low rates on account of A Century of Progress and have not entered into the convention fare certificate plan as outlined above for the eastern states. Ticket agents should be consulted with regard to these special rates.

Also, the railways in the eastern and southeastern states and eastern provinces of Canada are offering round-trip tickets at reduced rates on account of A Century of Progress. Particulars as to these special rates may be obtained from ticket agents.

ANNUAL MEETING

The annual meeting of the College will convene in the ballroom of the Stevens Hotel at 2 o'clock Thursday afternoon. Reports on the activities of the College will be presented by the officers and chairmen of the standing committees, followed by the election of officers.

HEADQUARTERS—HOTELS

General headquarters for the Clinical Congress will be established at the Stevens Hotel, located on Michigan Avenue between Seventh and Eighth Streets. This hotel affords unusual facilities for all activities of the Congress, as will be remembered by those who attended the Congress in

Chicago in 1929. The grand ballroom on the second floor with other large rooms on the third floor and the exhibition hall have been reserved for the exclusive use of the Congress. All of the evening sessions, the hospital conference on Monday, the annual meeting, the cancer and fracture symposia will be held in the grand ballroom. The registration and information bureau, together with the bulletin boards on which will be displayed the daily clinical program will be established in the exhibition hall together with the Technical Exhibition.

Chicago has many fine, large hotels, several within walking distance of the headquarters hotel. A list of the hotels recommended by the Committee on Arrangements is presented herewith. While Chicago's hotel facilities are very great and there should be no difficulty in securing first-class hotel accommodations, it is necessary for those who expect to attend the Clinical Congress to reserve their hotel accommodations immediately, as A Century of Progress Exposition is attracting to Chicago a very large number of visitors.

ADVANCE REGISTRATION

The hospitals of Chicago afford accommodations for a large number of visiting surgeons, but to insure against overcrowding, the attendance will be limited to a number that can be comfortably accommodated at the clinics—the limit of attendance being based upon the results of a survey of the amphitheaters, operating rooms, and laboratories of the hospitals and medical schools to determine their capacity for visitors.

CHICAGO HOTELS AND THEIR RATES

	Minimum Rates With Bath	
	Single	Double
Ambassador, North State Street at Goethe	$3.50	$6.00
Auditorium, Michigan Blvd. and Congress	3.50	6.00
Belden Stratford, 2300 Lincoln Park West	4.00	6.00
Belmont, Sheridan Road at Belmont	4.00	5.00
Bismarck, Randolph at LaSalle St	3.50	5.00
Blackstone, Michigan Blvd. and 7th St	3.00	5.00
Brevoort, 120 West Madison St	2.50	3.50
Congress, Michigan Blvd. and Congress	4.00	6.00
Drake, Lake Shore Drive and Michigan	3.00	5.00
Edgewater Beach, 5300 Sheridan Road	4.00	6.00
Great Northern, Jackson and Dearborn	2.50	4.00
Knickerbocker, 163 East Walton	3.00	5.00
LaSalle, LaSalle at Madison St	2.50	4.00
Morrison, 79 West Madison St	3.00	4.50
Palmer House, State and Monroe Sts	3.50	6.00
Pearson, 190 East Pearson St	3.00	5.00
Stevens, Michigan Blvd. bet. 7th and 8th	3.50	5.00

Attendance at all clinics and demonstrations will be controlled by means of special clinic tickets, which plan provides an efficient means for the distribution of the visiting surgeons among the several clinics and insures against overcrowding, as the number of tickets issued for each clinic will be limited to the capacity of the room in which that clinic will be given.

A registration fee of $5.00 is required of each surgeon attending the annual Clinical Congress, such fees providing the funds with which to meet the expenses of the meeting. To each surgeon registering in advance a formal receipt for the registration fee is issued, which receipt is to be exchanged for a general admission card upon his registration at headquarters. This card must be presented in order to secure clinic tickets and admission to the evening meetings.

SYMPOSIUM: CANCER IS CURABLE

Wednesday, 2:30 P.M.—Ballroom, Stevens Hotel

ROBERT B. GREENOUGH, M.D., Boston, Chairman, Committee on the Treatment of Malignant Diseases, Presiding.

General Subject of Curability of Cancer: FRANKLIN H. MARTIN, M.D., Director-General.

Cancer as an Arrestable Disease: CHARLES A. DUKES, M.D, Oakland, Calif.

General Cases of Five-year Cures: IRVIN ABELL, M.D., Louisville, Ky.; FRANK K. BOLAND, M.D., Atlanta, Ga.; JOHN JOSEPH GALLIGAN, M.D., Salt Lake City, Utah; CHEVALIER JACKSON, M.D., Philadelphia; ALSON R. KILGORE, M.D., San Francisco; CHARLES C. LUND, M.D., Boston; DAMON B. PFEIFFER, M.D., Philadelphia; EUGENE H. POOL, M.D., and JOHN A. VIETOR, M.D., New York; H. BECKWITH WHITEHOUSE, M.S., F.R.C.S., Birmingham, England.

Cancer of the Breast: MALVERN B. CLOPTON, M.D., St. Louis; E. STARR JUDD, M.D., Rochester, Minn.; JAMES MONROE MASON, M.D., Birmingham, Ala.; JOHN T. MOORE, M.D., Houston, Tex.; RICHARD R. SMITH, M.D., Grand Rapids, Mich.

Cancer of the Pelvic Organs and Breast: BROOKE M. ANSPACH, M.D., Philadelphia; HARRY S. CROSSEN, M.D., St. Louis; WILLIAM P. HEALY, M.D, New York; CHARLES C. NORRIS, M.D., Philadelphia.

Cancer of the Pelvic Organs: JAMES C. MASSON, M.D., Rochester, Minn.; CARL HENRY DAVIS, M.D., Milwaukee.

Cancer of the Cervix: GEORGE GELLHORN, M.D., St. Louis.

Cancer of the Kidney, Bladder, and Prostate: HERMAN L. KRETSCHMER, M.D., Chicago.

Cancer of the Rectum: ROBERT C. COFFEY, M.D., Portland, Ore.

Cancer of the Thyroid Gland and Large Intestine: JOHN DEJ. PEMBERTON, M.D., Rochester, Minn.

Cancer of the Thyroid: MARTIN B. TINKER, M.D., Ithaca, N.Y.

Malignant Bone Tumors: WILLIAM B. COLEY, M.D., New York.

CONFERENCES—SYMPOSIA—PUBLIC MEETINGS

SYMPOSIUM: TREATMENT OF FRACTURES

Tuesday, 2:30 P.M.—Ballroom, Stevens Hotel

CHARLES L. SCUDDER, M.D., Boston: Accomplishments and Ideals of the Regional Fracture Committees.

ROBERT H. KENNEDY, M.D., New York: Transportation of Early Long Bone Fractures; Co-ordination of the Activities of the Committee on the Treatment of Fractures of the American College of Surgeons with (a) the Red Cross, (b) the railroad association, (c) ambulances and morticians.

WILLIAM J. ESTES, JR., M.D., Bethlehem, Pa.: The After-Care in Preventing Disabilities Following Fractures.

ISIDORE COHN, M.D., New Orleans: Clinical Examination versus X-ray Examination in Fractures During Childhood.

FREDERICK J. TEES, M.D., Montreal: Dislocation of the Radiocarpal Joint.

ARTHUR STEINDLER, M.D., Iowa City, Iowa: Fracture Disabilities of the Wrist and Fingers.

COMMUNITY HEALTH MEETING

Wednesday, 8:00 P.M.—Chicago Stadium

At 7:30—Organ prelude.

Chorus of 1000 nurses in uniform.

Addresses of Welcome. PHILIP H. KREUSCHER, M.D., Chairman of Committee on Arrangements, and AUSTIN A. HAYDEN, M.D., President of Chicago Medical Society.

WILLIAM D. HAGGARD, M.D., Nashville, President, American College of Surgeons, Presiding.

Invocation. DR. LOUIS L. MANN, Rabbi, Chicago Sinai Congregation.

The American College of Surgeons—Its Message for You. FRANKLIN H. MARTIN, M.D., Chicago, Director-General.

Adding Years to Your Life and Life to Your Years. CHARLES H. MAYO, M.D., Rochester, Minn.

Cancer—It is a Curable Disease. BURTON J. LEE, M.D., New York.

A Century of Progress in Scientific Medicine. GEORGE W. CRILE, M.D., Cleveland.

The Care of the Prospective Mother. C. JEFF MILLER, M.D., New Orleans

Why Are You Nervous? ALFRED W. ADSON, M.D., Rochester, Minn.

Saving Your Eyesight. JOHN O. MCREYNOLDS, M.D., Dallas, Texas.

Doctors, Hospitals, and Patients. ROBERT JOLLY, Houston, Texas.

SYMPOSIUM ON UROLOGICAL SURGERY

Friday, 11:00 A.M.—Ballroom, Stevens Hotel

JOHN R. CAULK, M.D., St. Louis: Transurethral Surgery.

FRANK HINMAN, M.D., San Francisco: The Pathogenesis of Hydronephrosis.

JOSEPH F. MCCARTHY, M.D., New York: The Prostate Gland—Its Place in General Medicine; Newer Conception of Diagnosis and Therapy.

Discussion: HERMAN L. KRETSCHMER, M.D., and CHARLES MORGAN MCKENNA, M.D., Chicago.

INDUSTRIAL MEDICINE, TRAUMATIC SURGERY

Friday, 2:00 P.M.—Ballroom, Stevens Hotel

FREDERIC A. BESLEY, M.D., Chairman, Board on Industrial Medicine and Traumatic Surgery, Presiding.

Will Industrial Medicine and Traumatic Surgery Have a Place in the National Industrial Recovery Act? FRANKLIN H. MARTIN, M.D., Chicago.

A Three Years' Survey of Medicine and Surgery in Industry; Legislative Enactments and Other Indemnity Problems. M. N. NEWQUIST, M.D., Chicago.

Preventive Health Measures in Industry as Revealed in a Three Years' Survey. E. W. WILLIAMSON, M.D., Chicago.

Injuries of the Chest. CARL A. HEDBLOM, M.D., Chicago.

Acute Injuries of the Abdomen. WILLIAM O'NEILL SHERMAN, M.D., Pittsburgh.

Trauma of the Spinal Column. FRED H. ALBEE, M.D., New York.

Cerebral Injuries. CHARLES BAGLEY, JR., M.D., Baltimore.

Rehabilitation of the Injured. FREDERICK J. GAENSLEN, M.D., Milwaukee.

Co-operation of the Chief Surgeons with the Standardization of Medical Service in Industry. JOHN R. NILSSON, M.D., Omaha, President of the Association.

"A CENTURY OF PROGRESS"

Sunday, 7:00 P.M.—Court of the Hall of Science

Arcturus Ceremony.

7:20–8:15 P.M.

ROBERT JOLLY, Houston, Texas, Master of Ceremonies.

Music—St. Luke's Hospital Doctors' Alumnæ Association Orchestra.

Chorus—1000 Nurses in Uniform, Chicago Hospital Association.

8:15–10:00 P.M.

J. BENTLEY SQUIER, M.D., New York, President, American College of Surgeons, Presiding.

Address of Welcome. RUFUS DAWES, Chicago, President, "A Century of Progress."

Address of Welcome. WILLIAM ALLAN PUSEY, M.D., Chicago. Member, Executive Committee, and Chairman, Section on Medical Sciences, "A Century of Progress."

Response to Address of Welcome and Appreciation of What "A Century of Progress" Has Done for Scientific Medicine. GEORGE W. CRILE, M.D., Cleveland, Chairman, Board of Regents.

The American College of Surgeons and the Clinical Congress. Introduction of Representatives from Other Countries. FRANKLIN H. MARTIN, M.D., Chicago, Director-General, American College of Surgeons.

Greetings from Canada, Central America, South America, British Isles, Europe, and Australasia.

Surgery in the Past One Hundred Years. WILLIAM D. HAGGARD, M.D., Nashville, Tenn.

Surgery in the Next One Hundred Years. CHARLES H. MAYO, M.D., Rochester, Minn.

If You Were Sick—One Hundred Years Ago and Today. ROBERT JOLLY, Houston, Texas.

PROGRAMS FOR EVENING MEETINGS

BALLROOM OF THE STEVENS HOTEL AT 8:15

Presidential Meeting, Monday, October 9

Address of Welcome. PHILIP H. KREUSCHER, M.D., Chairman of Committee on Arrangements.
Introduction of Foreign Guests. FRANKLIN H. MARTIN, M.D., Director-General.
Address of Retiring President: The Hippocratic Code and the New Deal. J. BENTLEY SQUIER, M.D. New York.
Inauguration of Officers.
Inaugural Address: Surgery, the Queen of the Arts. WILLIAM D. HAGGARD, M.D., Nashville, Tenn.
John B. Murphy Oration in Surgery: The Story of a Master Surgeon. LOYAL DAVIS, M.D., Chicago.

Tuesday, October 10

The Common Syndrome of Rupture, Dislocation and Elongation of the Biceps Brachii; an Analysis of Fifty Cases. EDGAR L. GILCREEST, M.D., San Francisco.
Discussion: KELLOGG SPEED, M.D., and PAUL B. MAGNUSON, M.D., Chicago.
Fracture Oration: The Treatment of Fractures Involving Joints. W. E. GALLIE, M.D., F.R.C.S. (Eng.), Toronto, Ontario.
Technique and Results of the Operative Lengthening of the Femur. VITTORIO PUTTI, Bologna, Italy.
Discussion: WILLIAM R. CUBBINS, M.D., and BEVERIDGE H. MOORE, M.D., Chicago.

Wednesday, October 11

Sympathectomy in Children. DAVID EDWIN ROBERTSON, M.D., Toronto, Ontario.
Discussion: EDWIN W. RYERSON, M.D., Chicago.
Symposium on Vascular Diseases:
Thrombo-Angiitis Obliterans (Buerger's Disease). GEORGE E. BROWN, M.D., Rochester, Minn.
Ligation of Large Arteries. MONT ROGERS REID, M.D., Cincinnati.
Discussion: R. W. MCNEALY, M.D., and GEZA DE TAKATS, M.D., Chicago.
Mastopathia and Chronic Mastitis. H. BECKWITH WHITEHOUSE, M.S., F.R.C.S., Birmingham, England.
Discussion: MAX CUTLER, M.D., Chicago.

Thursday, October 12

Symposium on Diseases of the Thyroid:
Hyperthyroidism and Associated Diseases. GEORGE W. CRILE, M.D., Cleveland.
The Treatment of Exophthalmos. HOWARD C. NAFFZIGER, M.D., San Francisco.
Tumors of the Parathyroid Glands. EDWARD D. CHURCHILL, M.D., Boston.
Discussion: HARRY M. RICHTER, M.D., and LESTER DRAGSTEDT, M.D., Chicago.

Convocation—Friday, October 13

Invocation.
Conferring of Fellowships
Conferring of Honorary Fellowships.
Presidential Address: Surgeon of the Wilderness—Ephraim McDowell. WILLIAM D. HAGGARD, M.D., Nashville, Tenn.
Fellowship Address. ROBERT MAYNARD HUTCHINS, A.M., LL.D., President, University of Chicago.

SECTION ON OTOLARYNGOLOGY

Tuesday, 8:15 P.M.—North Ballroom, Stevens Hotel

Otolaryngology's Present-Day Economics. BURT R. SHURLY, M.D., Detroit, Michigan.
Discussion opened by ROBERT SONNENSCHEIN, M.D., and AUSTIN A. HAYDEN, M.D., Chicago.

SECTION ON OPHTHALMOLOGY

Thursday, 8:15 P.M.—North Ballroom, Stevens Hotel

Removal of Orbital Tumors. W. L. BENEDICT, M.D., Rochester, Minn.
Discussion opened by RICHARD GAMBLE, M.D., Chicago.
Surgical Correction of Ocular Disfigurement. MEYER WIENER, M.D., St. Louis.
Discussion opened by SAMUEL J. MEYER, M.D., Chicago.

ANNUAL HOSPITAL STANDARDIZATION CONFERENCE

Monday, 10:00–12:30—Ballroom, Stevens Hotel

J. BENTLEY SQUIER, M.D., New York, President, American College of Surgeons, presiding.

Address of Welcome: J. BENTLEY SQUIER, M.D., New York.

The 1933 Hospital Standardization Survey and Announcement of List of Approved Hospitals: FRANKLIN H. MARTIN, M.D., Chicago; Director-General, American College of Surgeons.

The Hospital Standardization Movement in Relation to the Practice of Internal Medicine: WALTER L. BIERRING, M.D., Des Moines, Iowa.

Opportunities of the Surgeon and the Hospital in Promoting Community Interest in the Proper Care of the Sick and Injured: BERT W. CALDWELL, M.D., Chicago.

Preparation for a Surgical Career: WILLIAM D. HAGGARD, M.D., Nashville, Tenn.

The Modern Philosophy of Medicine: REV. ALPHONSE M. SCHWITALLA, S.J., Ph.D., St. Louis.

A Century of Progress: EBEN J. CAREY, M.D., Milwaukee, Wis.

The Next Century of Progress in Medicine: GEORGE W. CRILE, M.D., Cleveland.

Motion picture (sound), "Good Hospital Care."

Monday, 2:00–5:00—Ballroom, Stevens Hotel

ROBERT B. GREENOUGH, M.D., Boston, presiding.

Round Table Conference: Medical and Hospital Economics—Maintaining as low hospital charges as are consistent with good care of the patient—from the standpoint of:

- The Surgeon: ALEXANDER W. BLAIN, M.D., Detroit, Mich.
- The Internist: S. MARX WHITE, M.D., Minneapolis, Minn.
- The Specialist: AUSTIN A. HAYDEN, M.D., Chicago.
- The Radiologist: H. B. PODLASKY, M.D., Milwaukee, Wis.
- The Pathologist: J. J. MOORE, M.D., Chicago.
- The Hospital Management: PAUL H. FESLER, Chicago.

Hospital Economics as Applied to the Small Hospital: CLINTON F. SMITH, Waterloo, Iowa.

Prepayment Plans for Hospital Service: WILLIAM H. WALSH, M.D., Chicago.

The Alameda Plan: CHARLES A. DUKES, M.D., Oakland, Calif.

Tuesday, 9:30–12:30—Ballroom, Stevens Hotel

ALEXANDER R. MUNROE, M.D., Edmonton, Alberta, presiding.

The Application of Hospital Standardization in the Small Hospital: MARSH T. LEWIS, Princeton, Ind.

The Hospital Annual Report: CHARLES E. REMY, M.D., Minneapolis, Minn.

Convalescent Care for the Patient: G. HARVEY AGNEW, M.D., Toronto, Ontario.

The Organization, Management and Functioning of the Department of Anæsthesia in a 200 Bed Hospital: BEVERLY LEECH, M.D., Regina, Saskatchewan.

The Organization, Management, and Functioning of the Clinical Laboratory: ROBERT I. GLENN, M.D., Oakland, Calif.

Clinical and Clinico-Pathologic Conferences: OLIVER W. LOHR, M.D., Saginaw, Mich.

Tuesday, 2:00–5:00—St. Luke's Hospital

Demonstration in hospital administration conducted by CHARLES A. WORDELL, Manager, St. Luke's Hospital. Admitting and discharging patients. Care of emergencies in hospitals. Nursing administration and service. Business methods.

Wednesday, 9:30–12:30—Ballroom, Stevens Hotel

Joint Conference—American College of Surgeons and Association of Record Librarians of North America: R. C. BUERKI, M.D., Madison, Wis., presiding.

Plan and Scope of the Record Department: MARY M. NEWTON, Pittsburgh, Pa.

A Survey of Cancer Records in Hospitals: PRISCILLA WEIR, New York.

The Importance of Accurate and Complete Records on Fracture Cases: FRANK D. DICKSON, M.D., Kansas City, Mo.

The Importance of Accurate and Complete Obstetrical Records: JOHN R. FRASER, M.D., Montreal, Quebec.

Round table conference—problems associated with the obtaining of good clinical records in hospitals.

Wednesday, 2:00–5:00—Albert Merritt Billings Hospital

Demonstration in hospital administration conducted by JOHN C. DINSMORE, Superintendent, University of Chicago Clinics. Organization and management of the clinical record department. Organization and functioning of the social service department. Organization and management of the obstetrical department. Operating room management and procedure. Tour of observation including the Chicago Lying-In Hospital.

Thursday, 9:30–12:30—Ballroom, Stevens Hotel

Round table conference—administrative, medical, nursing, economic, and social problems affecting hospitals. Conducted by ROBERT JOLLY, Houston, Texas, and R. C. BUERKI, M.D., Madison, Wis.

Thursday, 2:00–5:00—Local Hospitals

Demonstrations of hospital administration in approved Chicago hospitals.

PRELIMINARY CLINICAL PROGRAM

GENERAL SURGERY, GYNECOLOGY, OBSTETRICS, ORTHOPEDICS, UROLOGY, PROCTOLOGY, SURGICAL PATHOLOGY, ETC.

PASSAVANT MEMORIAL HOSPITAL—NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

Tuesday

LEANDER W. RIBA—9. The use of the electro-urethrotome in urethral strictures.

ARTHUR H. CURTIS and GEORGE H. GARDNER—9. Gynecological operations.

JOHN A. WOLFER—9. Cholecystitis. carcinoma of colon.

JACOB R. BUCHBINDER—9. Thyroid surgery.

JOHN S. COULTER and staff—10. Hyperpyrexia by physical agents in the treatment of arthritis.

RUDOLPH W. HOLMES and staff—2. Symposium on cardiac diseases in their obstetric associations. CHAUNCEY C. MAHER: Etiology and pathology. JAMES E. FITZGERALD: Medical aspects and treatment. JAMES H. BLOOMFIELD: Obstetrical aspects and treatment.

JOHN A. WOLFER—2. Dry clinic: Alimentation of the critically ill patient by jejunal feedings.

LOYAL DAVIS, HALE HAVEN and DAVID A. CLEVELAND—2. Presentation and review of neurosurgical cases.

Wednesday

HARRY M. RICHTER—9. Thyroid surgery.

LOYAL DAVIS—9. Neurological surgery.

SUMNER L. KOCH and MICHAEL L. MASON—9. Nerve and tendon surgery of the hand.

JAMES T. CASE—10. Roentgenology.

PHILIP H. KREUSCHER—2. Hip joint surgery.

ALLEN B. KANAVEL, SUMNER L. KOCH and M. L. MASON—2. Review of twenty years of surgery of the hand.

PAUL B. MAGNUSON—2. Ununited fracture of the neck of the femur, bone graft in the spine.

RUDOLPH W. HOLMES and staff—2. Symposium on toxæmias of late pregnancy, renal and hepatic. JAMES P. SIMONDS: Etiology and pathology. CHESTER C. DOHERTY: Symptoms and laboratory investigation. DAVID S. HILLIS: Medical (expectant) treatment. RUDOLPH W. HOLMES: Obstetrical treatment.

LEANDER W. RIBA—2. Dry clinic: Prostatic resection.

EMIL D. W. HAUSER—2. Orthopedic surgery.

Thursday

ARTHUR H. CURTIS and GEORGE H. GARDNER—9. Gynecological operations.

JOHN A. WOLFER—9. Cholecystitis; carcinoma of the breast.

JACOB R. BUCHBINDER—9. Abdominal surgery.

JOHN S. COULTER and staff—10. Physical therapy in the after-treatment of hand injuries.

PHILIP H. KREUSCHER—2. Shoulder and knee-joint derangement.

RUDOLPH W. HOLMES and staff—2. Symposium on obstetrical hæmorrhages. RUDOLPH W. HOLMES: Ablatio placentæ. DAVID S. HILLIS: Placenta prævia. MAGNUS P. URNES: Postpartum hæmorrhages. THEODORE W. BLUECHEL: Treatment of sequential anæmias.

CHARLES A. ELLIOTT, WALTER H. NADLER, PAUL STARR, M. HERBERT BARKER, HOWARD B. CARROLL and HOWARD L. ALT—2. Symposium on hepatic disease.

Friday

HARRY M. RICHTER—9. Gastric surgery.

LOYAL DAVIS—9. Neurologic surgery.

SUMNER L. KOCH and MICHAEL L. MASON—9. Irradiation ulcers of the hand, Dupuytren's contracture.

JAMES T. CASE—10. Roentgenology.

JOHN S. COULTER and staff—10. Physical therapy in arthritis.

PAUL B. MAGNUSON—2. Demonstration of principles for overcoming deformity in ununited fractures before operation; bone grafts for ununited fractures.

RUDOLPH W. HOLMES and staff—2. Symposium on hyperemesis gravidarum. CHESTER C. DOHERTY: Etiology and pathology. MAGNUS P. URNES: Symptoms and clinical course. JAMES H. BLOOMFIELD: Treatment.

Staff—2. Symposium on gastric ulcer. HARRY M. RICHTER: Surgical aspects. ANDREW C. IVY, G. B. FAULEY and J. R. ORNDORFF: Results of the use of gastric mucin in peptic ulcer experimentally produced. SAMUEL J. FOGELSON and A. J. ATKINSON: Use of gastric mucin in treatment of gastric ulcer.

ST. LUKE'S HOSPITAL

Monday

H. E. MOCK, A. REID MORROW and CHARLES SHANNON—2. General surgical operations.

E. OLDBERG—2. Neurological surgery.

Tuesday

H. O. JONES, WILLIAM P. CARLISLE, M. J. KILEY, E. A. EDWARDS and JOHN BREWER—9. Gynecological operations; early human embryo, demonstration.

CARL HEDBLOM and WILLARD VAN HAZEL—9. Thoracic surgery.

H. E. MOCK—2. Reconstructive surgery.

L. L. MCARTHUR and S. W. MCARTHUR—2. General surgery.

Wednesday

L. E. SCHMIDT—9. Urological clinic.

E. W. RYERSON and F. A. CHANDLER—9. Orthopedic operations.

S. C. PLUMMER—9. General surgery.

H. E. JONES and T. L. HANSEN—9. General surgery.

E. W. RYERSON, R. O. RITTER and H. O. SOFIELD—2. Orthopedic operations.

FRANK E. DAVID, C. J. DEBERE and G. V. PONTIUS—2. Rectal surgery.

Thursday

G. DETAKATS—9. Surgery in juvenile diabetes; ambulatory vein ligation of varicose veins.

H. E. MOCK—9. General surgery.

HARRY CULVER—9. Urological clinic.

H. E. MOCK, A. REID MORROW and CHARLES SHANNON—2. Skull fractures.

W. R. CUBBINS—2. General surgery.

H. B. THOMAS and F. W. HARK—2. Orthopedic clinic.

Friday

W. F. LYON—9. Dislocations of the shoulder with fracture of the greater trochanter.

H. POTTS and F. W. MERRIFIELD—9. Oral surgery, operative.

E. W. RYERSON, F. A. CHANDLER and R. O. RITTER—2 Orthopedic clinic.

PRESBYTERIAN HOSPITAL AND RUSH MEDICAL COLLEGE

Tuesday

A. D. BEVAN—9. Surgery of the breast.
V. C. DAVID—9. Carcinoma of sigmoid.
H. L. KRETSCHMER—9. Kidney surgery.
R. H. HERBST—9. Transurethral electro resection of prostate gland.
KELLOGG SPEED—9. Tumors of chest wall, demonstration of cases, lantern slides.
A. H. MONTGOMERY—11. Abdominal surgery in children.
A. VERBRUGGHEN—2. Neurosurgical operation.

Wednesday

A. D. BEVAN—9. Hernia and undescended testicle.
F. B. MOOREHEAD—9. Plastic surgery of mouth and face.
C. B. DAVIS—9. Tumors of the large intestine.
H. L. KRETSCHMER—9. Surgery of the bladder.
N. S. HEANEY—9. Vaginal surgery.
DR. GATEWOOD—10. Carcinoma of the stomach, follow-up clinic.
E. M. MILLER—10. Thyroid surgery.
H. A. OBERHELMAN—10. Surgery in diabetic patients.
E. R. MCCARTHY—11. Strangulated hernia in infants.
W. J. POTTS—12. Fracture problems.

Thursday

A. D. BEVAN—9. Surgery of gall bladder and bile tract.
H. L. KRETSCHMER—9. Transurethral resection of the prostate.
F. B. MOOREHEAD—9. Cleft palate surgery; operative treatment of ankylosis of jaw.
DR. GATEWOOD—9. Gastric resection for ulcer.
R. H. HERBST—9. Diverticula of urinary bladder.
R. H. HERBST and C. W. APFELBACH—9. Unusual urinary anomalies.
Staff—9. Dry clinic: E. D. ALLEN, endometriosis; C. P. BAUER, dystocia; AARON KANTER, recognition of early carcinoma of uterus.
G. L. MCWHORTER—10. Fracture of the greater tuberosity of the humerus.
A. VERBRUGGHEN—10. Spinal cord injuries.

Friday

Staff—9. Dry clinic: A. D. BEVAN. Present status of anæsthesia. H. L. KRETSCHMER. Genito-urinary surgery. R. C. BROWN. Treatment of massive hæmorrhage in gastric ulcer. V. C. DAVID. Significance of polyps of large bowel. E. M. MILLER. Method of intravenous injection over long period of time. R. H. HERBST. Fibrosis of bladder neck. F. H. STRAUS. Obstructive jaundice. G. L. MCWHORTER. Reconstruction of common bile duct, cases. M. L. LORING. Granuloma inguinale, cases. S. E. LAWTON. Cholecystenterostomy, indications.
E. J. BERKHEISER—2. Orthopedic clinic.

ST. FRANCIS HOSPITAL

Thursday

E. FOWLER—2. Painful shoulder.
H. REINSCH—2. Indications for duodenal and jejunal drainage and feeding.
T. E. CONLEY—2. Value of hyperventilation, prevention and treatment of thrombophlebitis.
B. FILLIS—2. Mechanical aids in urology.
H. F. MARS—2. Uses and improved methods of administration of parenteral fluids.
I. H. CHILCOTT—2. Management of gastric, biliary and jejunal fistula.

MERCY HOSPITAL

Tuesday

E. M. BROWN—9. Malignancy of the colon.
J. E. KELLY—9. Chronic intestinal fistula; extensive ventral hernia.
GEORGE GRIFFIN—9. Pyloric obstruction.
J. D. CLARIDGE—9. Fractures and dislocations of the cervical spine.
C. J. LARKIN—9. Rupture of the spleen simulating acute appendicitis.

Wednesday

M. F. MCGUIRE—9. Biliary tract surgery.
C. F. SAWYER—9. Acute pancreatitis; perforating gastric and duodenal ulcers.
C. L. MARTIN—9. Anal fistulectomies in cases with pulmonary tuberculosis.
L. E. GARRISON—9. Carcinoma of the colon; carcinoma of the breast.
HERBERT E. LANDES—9. Surgical anatomy of vesical orifice and urethral obstructions; treatment of bladder tumors.

Thursday

L. D. MOORHEAD—9. Toxic goiters; differential diagnosis of cases of dysthyroidism and hyperthyroidism with indication for operation and management.
W. J. PICKETT—9. Technical considerations in posterior gastro-enterostomy.
F. E. PIERCE—9. Fracture cases.
F. M. DRENNAN and F. C. VALDEZ—9. Gastro-intestinal clinic.

Friday

HENRY SCHMITZ and HERBERT E. SCHMITZ—9. Gynecological clinic; surgery and radiation therapy.
JOSEPH LAIBE—9. Carcinoma of the genito-urinary tract.
A. M. VAUGHN—9. Cystic hygroma in an infant.

CHILDREN'S MEMORIAL HOSPITAL

Monday

FREMONT A. CHANDLER, FERDINAND SEIDLER and CHARLES N. PEASE—2. Osteochondroma of fibula; radio-ulnar synostosis; spastic paralysis; Thomsen's disease; hip fusion; knee fusion; spine fusion; flat feet; congenital dislocation of hip; synovectomy in arthritis; scoliosis.

Tuesday

FREMONT A. CHANDLER, CHARLES N. PEASE and FERDINAND SEIDLER—9. Spine fusion; hip fusion; knee fusion; section of obturator nerves in spastic paralysis; congenital dislocation of hip.
FREDERICK B. MOOREHEAD—2. Cleft lip and cleft palate operations; demonstration of operated cases of ankylosis of the jaw.

Wednesday

JOHN GRAHAM—9. General surgical operations and demonstrations.

Thursday

HERMAN L. KRETSCHMER—9. Urological operations.
C. A. ALDRICH—9. Clinical consideration of urological conditions.
W. G. HIBBS—9. Pathological demonstration of urological cases.

Friday

ALBERT H. MONTGOMERY and J. J. MUSSIL—9. Appendicitis in children; lymphangioma of the omentum; sacral chordomas; fractures of the skull and extremities in children.
STANLEY LAWTON—9. Undescended testicle.

COOK COUNTY HOSPITAL

Monday

SUMNER L. KOCH—2. General surgery.
F. H. FALLS—2. Gynecology.
E. J. BERKHEISER—2. Orthopedics.
WILLIAM R. CUBBINS—2. General surgery.
MARSHALL DAVISON—2. General surgery.

Tuesday

SUMNER L. KOCH—9. Diagnostic clinic.
R. W. MCNEALY—9. General surgery.
AARON KANTER—9. Gynecology.
GEORGE DAVIS—9. General surgery.
A. H. MONTGOMERY—9. General surgery.
A. H. CONLEY—9. Orthopedics.
CAREY CULBERTSON—9. Gynecology.
J. O'DONOGHUE—9. General surgery.
HARRY CULVER—9. Urology.
H. JACKSON—9. General surgery.
MARCUS HOBART—9. Orthopedics.
VERNON C. DAVID—9. Diagnostic clinic.
DR. GATEWOOD—9. General surgery.
J. P. GREENHILL—2. Gynecology.
RALPH B. BETTMAN—2. Surgery in tuberculosis.
E. WARSZEWSKI—2. General surgery.

Wednesday

CHANNING BARRETT—9. Gynecology.
HARRY CULVER—9. Urology.
V. L. SCHRAGER—9. General surgery.
GEORGE APFELBACH—9. General surgery.
J. G. FROST—9. General surgery.
R. C. SULLIVAN—9. General surgery.
L. L. VESEEN—9. Urology.
FRANK JIRKA—9. General surgery.
R. VAUGHAN—9. General surgery.
PHILIP H. KREUSCHER—9. Orthopedics.
CHARLES M. MCKENNA—9. Urology.
H. ROLNICK—2. Urology.
HARRY CULVER—2. Urology.
GEORGE DAVIS—2. General surgery.
J. R. BUCHBINDER—2. General surgery.
DAVID HILLIS—2. Obstetrical operations.
SUMNER L. KOCH—2. General surgery.

Thursday

PHILIP H. KREUSCHER—9. Orthopedics.
CHANNING BARRETT—9. Gynecology.
GEORGE DAVIS—9. General surgery.
R. W. MCNEALY—9. General surgery.
MARCUS HOBART—9. Orthopedics.
D. HORNER—9. Gynecology.
KARL A. MEYER—9. General surgery.
E. W. FISCHMAN—9. Gynecology.
A. H. MONTGOMERY—9. General surgery.
MAX THOREK—9. General surgery.
A. H. CONLEY—9. Orthopedics.
D. H. LEVINTHAL—9. Orthopedics.
JOHN HARGER—2. General surgery.
F. H. FALLS—2. Gynecology.
E. J. BERKHEISER—2. Orthopedics.
RALPH BETTMAN—2. General thoracic surgery.
WILLIAM R. CUBBINS—2. General surgery.

Friday

GEORGE APFELBACH—9. General surgery.
AARON KANTER—9. Gynecology.
R. C. SULLIVAN—9. General surgery.
CAREY CULBERTSON—9. Gynecology.
VERNON C. DAVID—9. General surgery.
MARCUS HOBART—9. Orthopedics.
F. G. DYAS—9. General surgery.
J. O'DONOGHUE—9. General surgery.
H. JACKSON—9. General surgery.
DR. GATEWOOD—9. General surgery.
JOHN HARGER—2. General surgery.
J. R. BUCHBINDER—2. General surgery.
MARSHALL DAVISON—2. General surgery.
E. WARSZEWSKI—2. General surgery.
SUMNER L. KOCH—2. General surgery.

INSTITUTE OF TRAUMATIC SURGERY

(St. Luke's Hospital)

Wednesday

GEORGE G. DAVIS—9. Rupture of the urethra.
JOHN D. ELLIS—9:15. Routine examination of injured back.
FREMONT A. CHANDLER—9:30. Separation of isthmus of lower lumbar vertebræ.
WILLIAM R. CUBBINS—9:45. Old dislocation of shoulder.
HARRY E. MOCK—10. Demonstration of cases of multiple injury.
LEROY P. KUHN—10:15. Ruptured spleen and other abdominal cases.
E. W. RYERSON—10:30. Spondylolisthesis in relation to injuries.
GEORGE L. APFELBACH—10:45. Cotton's fracture.
R. W. MCNEALY—11. Immediate repair of injured blood vessels.
E. C. HOLMBLAD—11:15. Compression fractures of the spine.
PHILIP H. KREUSCHER—11:30. Knee joint injuries.
HOLLIS E. POTTER—11:45. Some X-ray aspects of silicosis.
C. R. G. FORRESTER—12. Reduction of fractures under local anæsthesia together with ambulatory treatment, moving picture demonstration.
LEROY THOMPSON—1:30. Intra-ocular foreign bodies.
SYDNEY WALKER, JR.—1:45. Lacerated wounds of the eyeball.
PAUL B. MAGNUSON—2. Anomalies of the spine.
A. M. HARVEY—2:15. Demonstration of rehabilitated cases.
EDSON B. FOWLER—2:30. Troublesome shoulders.
KELLOGG SPEED—2:45. Injuries to internal semilunar cartilage.
HERMAN L. KRETSCHMER—3. Management of traumatised kidney.
JAMES A. VALENTINE—3:15. Treatment of ruptured biceps tendon.
FRED M. MILLER—3:30. The injured hand.
SIDNEY B. MACLEOD—3:45. New apparatus to increase efficiency of Thomas splint.
FRED W. SLOBE—4. Bilateral renal carbuncle with perinephritic abscess.
HART E. FISHER—4:15. Electric burns in children.
CLARENCE W. HOPKINS—4:30. Modern treatment of compression fractures of the spine.
ARNO B. LUCKHARDT—4:45. Choice of anæsthetic in surgical shock.

MOTHER CABRINI MEMORIAL HOSPITAL

Tuesday

EUGENE J. CHESROW—9. General surgical clinic.

Wednesday

EUGENE J. CHESROW—9. General surgical clinic.

Thursday

EUGENE J. CHESROW—9. General surgical clinic.

MICHAEL REESE HOSPITAL

Tuesday

ALFRED A. STRAUSS, SIEGFRIED F. STRAUSS, JAMES PATEDJL and ROBERT A. CRAWFORD. Stomach resections for gastric and duodenal ulcer; common duct duodenal anastomosis and gastro-enterostomy for chronic obstructive jaundice.

GEORGE L. DAVENPORT and RALPH BETTMAN. Gall-bladder surgery; surgery of the common duct.

D. C. STRAUSS. Thyroid surgery.

E. FRIEND. General surgery; surgery of the gall bladder.

BERNARD PORTIS. Thyroid surgery; surgery of the rectum.

HARRY RICHTER. Thyroid surgery; gall-bladder surgery.

MAX CUTLER. Surgery of the breast.

GUSTAV KOLISCHER. Diathermy of bladder tumor; nephrectomy for tuberculosis.

IRVING KOLL. Electrical resection of prostate; nephrolithotomy.

DANIEL H. LEVINTHAL. Internal derangements of the knee joint; removal of semi-lunar cartilage; synovectomy for chronic arthritis; bone lengthening operation.

JULIUS E. LACKNER. Abdominal hysterectomy; interposition operation; rectovaginal fistula.

JOSEPH L. BAER and RALPH REIS. Complete perineal laceration, ovarian tumor and pelvic inflammation.

Wednesday

D. C. STRAUSS. Thyroid surgery; gall-bladder surgery.

RALPH BETTMAN. Surgery of the chest.

GEORGE L. DAVENPORT. General surgery.

ALFRED A. STRAUSS, SIEGFRIED F. STRAUSS and ROBERT A. CRAWFORD. Sectional colectomy for ulcerative colitis and pyloroplasty for congenital pyloric stenosis.

BERNARD PORTIS. General surgery and surgery of the colon.

MORRIS L. PARKER. General surgery.

JAMES PATEDJL. General surgery.

JOSEPH EISENSTAEDT. Undescended testis; suprapubic prostatectomy.

HARRY ROLNICK. Electric resection of prostate; pyelotomy for stones.

PHILIP LEWIN and SIDNEY SIDEMAN. Orthopedic clinic, shoulder, elbow, hand, hip, pelvis.

L. E. FRANKENTHAL, SR. and L. E. FRANKENTHAL, JR. Gynecological operations.

W. H. RUBOVITS. Obstetrical and gynecological clinic, demonstration of forceps, version and complete suture, episiotomy.

IRVING STEIN and M. L. LEVENTHAL. Obstetrical clinic, low cervical cæsarean under local anæsthesia.

Thursday

RALPH BETTMAN. Surgery of gall bladder and common duct.

ALFRED A. STRAUSS, SIEGFRIED F. STRAUSS and ROBERT A. CRAWFORD. Surgical diathermy for carcinoma of the rectum; resections for carcinoma of the stomach.

D. C. STRAUSS. Surgery of colon, small intestine, thyroid.

GEORGE L. DAVENPORT. Surgery of the common duct.

BERNARD PORTIS. General surgery.

SIEGFRIED F. STRAUSS. General surgery.

HARRY RICHTER. Surgery of the thyroid.

E. FRIEND. Surgery of the gall bladder and common duct.

ALFRED E. JONES. Nephrectomy for tuberculous kidney; suprapubic prostatectomy.

IRVING SHAPIRO. Diathermy of bladder tumor; nephrectomy for tumor of kidney.

DANIEL H. LEVINTHAL. Surgery of the spine, fusion operation for scoliosis and for tuberculosis.

CHARLES M. JACOBS. Orthopedic clinic.

JULIUS E. LACKNER. Gynecological operations.

JOSEPH L. BAER and RALPH REIS. Prolapse; vaginal hysterectomy; fibroids; occiput posterior.

Friday

ALFRED A. STRAUSS, SIEGFRIED F. STRAUSS, JAMES PATEDJL and ROBERT A. CRAWFORD. Subtotal gastrectomy for gastrojejunal ulcer; resection of colon for carcinoma.

D. C. STRAUSS. Surgery of the thyroid and general surgery.

GEORGE L. DAVENPORT and RALPH BETTMAN. Gall-bladder surgery and surgery of the common duct.

RALPH BETTMAN. Thoracic surgery.

BERNARD PORTIS. Surgery of the colon and rectum.

MORRIS L. PARKER. General surgery.

MAX CUTLER. Surgery of the breast; use of radiotherapy in carcinoma.

FREDERICK LIEBERTHAL. Suprapubic prostatectomy; ureterotomy.

J. S. GROVE. Undescended testes.

PHILIP LEWIN and SIDNEY SIDEMAN. Orthopedic clinic, back, hip, knee, foot, shoulder; demonstration of arthritis cases.

L. E. FRANKENTHAL, SR. and L. E. FRANKENTHAL, JR. Gynecological clinic.

W. H. RUBOVITS. Gynecological clinic.

IRVING STEIN and M. L. LEVENTHAL. Gynecological clinic.

ST. MARY OF NAZARETH HOSPITAL

Monday

A. S. SAMPOLINSKI—2. General surgical clinic.

E. H. WARSZEWSKI and P. F. CZWALINSKI—2. Inguinal hernia clinic.

THAD LARKOWSKI—2. Demonstration of blood transfusion.

Tuesday

GEORGE MUELLER—9. General surgical clinic.

S. R. PIETROWICZ—9. Spinal puncture and anæsthesia—indications, contra-indications, advantages, disadvantages, demonstrations.

C. C. BUCZYNSKI—2. Varicocele operations and demonstrations.

M. J. BADZMIEROWSKI and B. PIERZYNSKI—2. Goiter clinic, operations and demonstration of cases.

Wednesday

T. Z. XELOWSKI—9. Gynecology and abdominal surgery.

W. A. KUFLEWSKI—9. Emergency and general surgery.

F. DERENGONSKI—9. Surgical clinic.

THOMAS PLANT—9. General surgery.

A. A. THIEDA—9. General surgery.

FRANK TENZAR—9. General surgery.

JOHN TENZAR—9. General surgery.

CHESTER CHALLENGER—9. X-ray demonstration.

MICHAEL KUTZA—2. General surgery.

F. A. MACKOWIAK—2. General surgery.

M. E. UZNANSKI—2. Obstetrical clinic, low cæsarean section.

Thursday

LEO CZAJA—9. Orthopedic clinic; maggot treatment of osteomyelitis.

E. MACDONALD—9. Abdominal surgery.

H. H. HILL—9. Demonstration of pathological specimens.

A. V. PARTIPILLO—2. Aseptic resection of the bowel, demonstration of cases, moving picture exhibition.

M. E. UZNANSKI—2. Surgical anatomy of the perineum, lantern slide demonstration.

Friday

JOSEPH WELFELD—9. Urological clinic.

GEORGE MUELLER—9. General surgery.

CHESTER CHALLENGER—9. X-ray demonstration.

H. H. HILL—9. Demonstration of pathological specimens.

ROBERT R. FLANNERY—2. Gall-bladder surgery.

LEO P. KOZAKIEWICZ—2. Cæsarean section, indications, contra-indications, demonstrations.

MOUNT SINAI HOSPITAL

Tuesday

V. L. SCHRAGER and J. T. GAULT—9. Hernia, breast and biliary surgery.
ISRAEL DAVIDSOHN—11. Pathological demonstration.
M. I. KAPLAN—11. X-ray diagnosis and therapy.
GUSTAV KOLISCHER and HARRY ROLNICK—2. Genito-urinary surgery.

Wednesday

HARRY M. RICHTER, J. M. MORA and D. WILLIS—9. Gastric and thyroid surgery.
ISRAEL DAVIDSOHN—11. Pathological demonstration.
M. I. KAPLAN—11. X-ray diagnosis and therapy.
ALFRED A. STRAUSS, S. STRAUSS, E. GREENE, I. E. BISHKOW and B. SAYRE—2. Gastro-intestinal surgery.
RALPH B. BETTMAN and L. HANDELMAN—2. Intrathoracic surgery, operations.

Thursday

AARON KANTER, A. F. LASH, E. SCHWIED and H. L. KLAWANS—9. Gynecological operations.
ISRAEL DAVIDSOHN—11. Pathological demonstration.
M. I. KAPLAN—11. X-ray diagnosis and therapy.
CHARLES JACOBS and LEO MILLER—2. Orthopedic operations.

Friday

HARRY ROLNICK—9. Genito-urinary surgery.
ISRAEL DAVIDSOHN—11. Pathological demonstration.
M. I. KAPLAN—11. X-ray diagnosis and therapy.

Dry Clinics—Daily, 9 and 2

ISRAEL DAVIDSOHN. Value of biopsy in surgery.
HENRY BUXBAUM. Toxæmias of pregnancy.
GUSTAV KOLISCHER—Electrosurgery in cancer therapy.
AARON KANTER. Chorio-epithelioma following a vesicular mole; functional uterine hæmorrhage.
HARRY ROLNICK. Bladder tumors.
A. F. LASH. Treatment of birth injury; early diagnosis of uterine cancer.
DAVID A. WILLIS. Relation of adrenals to thyrotoxicosis; morbidity in operation for acute appendicitis in relation to the question of drainage; demonstration of a universal traction splint as used in a small hospital.
EMIL L. AISON. Fractures of the maxilla and mandible.
M. REESE GUTTMAN. Recent advances in the treatment of malignant diseases about the head and neck; endoscopic clinic.
Staff. Symposium: Cancer of the lung. I. M. TRACE, medical aspect; JACOB LIFSCHUTZ, bronchoscopic aspect; ISRAEL DAVIDSOHN, pathological aspect; M. I. KAPLAN, X-ray aspect.
MAURICE LEWISON. Medical appraisal of surgical risks.
JOSEPH T. GAULT. Present status of the treatment of varicose veins.
EARLE I. GREENE. Intestinal obstruction.
J. M. MORA. Inflammatory lesions of the thyroid.
I. E. BISHKOW. Present status of blood transfusion.
CHARLES JACOBS. Supracondyloid fractures of femur; a modified treatment of correction of hallux valgus.
EARL HERRON. A case of acute septic epiphysitis with interesting X-ray changes in the head of the femur; bridge cast in treatment of supracondylar fractures of the elbow joint.
LEO MILLER. Effect of radio-opaque substances on the synovial membrane of the knee joint; rheumatoid arthritis.

RAVENSWOOD HOSPITAL

Tuesday

G. W. GREEN—9. Gall-bladder surgery, mortality and morbidity.
C. A. BUSWELL—9:30. Survey of cancer study organization in a private hospital.
D. B. POND—10. Orthopedic surgery.
E. W. MUELLER and J. J. MOORE—10:30. Carcinoma of testis.
M. FIELD—11. Diagnosis and management of sterility.
L. C. FRENCH and D. L. JENKINSON—11:30. Gastric syphilis.

Wednesday

G. DE TARNOWSKY and J. J. MOORE—9. Carcinoma of colon, modified Kraske operation.
J. IRELAND—9:30. Fractures of the elbow.
R. F. WEISSBRENNER—10. Emotions as etiological factors in hyperthyroidism.
C. H. LOCKWOOD—10:15. Headaches.
H. P. SAUNDERS—11. Blood transfusion.
L. E. DAY—11:15. Obstetrics.
J. F. OATES—11:30. Spinal anæsthesia.

Thursday

C. C. RENTFRO—9. Obstetrical anæsthesia.
W. F. GROSVENOR—9:15. Cæsarean section.
A. C. HAMMETT—9:30. Mental disturbances of diabetics.
A. V. BERGQUIST—9:45. Indigestion.
F. N. BUSSEY—10. Granulosa cell carcinoma of ovary.
R. E. DYER—10:30. Surgical technique.
P. J. SARMA—11. Paramedian abdominal incision.
F. R. VON NAHOWSKI—11:15. Mortality in appendicitis.
E. B. WILLIAMS—11:30. Perthes' disease; fracture of spine.

JACKSON PARK HOSPITAL

Monday

F. L. BARBOUR—2. Dry clinic: Symposium on treatment of pulmonary tuberculosis, surgical and medical.

Tuesday

T. H. KELLEY—9. General surgical clinic.
ARRIE BAMBERGER—10. General surgical clinic.
C. C. CLARK—11. General surgical clinic.
S. B. MACLEOD—2. Fracture clinic.

Wednesday

ARRIE BAMBERGER—9. General surgical clinic.
H. HOYT COX—10. General surgical clinic.
S. W. MARCHMONT-ROBINSON—2. Dry clinic: Hand infections as related to industrial surgery.
H. F. SPIERLING—3. Mortality of appendicitis.

Thursday

ARRIE BAMBERGER—9. General surgical clinic.
T. H. KELLEY—10. General surgical clinic.
G. MARCHMONT-ROBINSON—11. Injection treatment of hæmorrhoids.
E. ALLEN PARSONS—12. Postoperative treatment of ruptured appendix with peritonitis.
R. T. FARLEY—1. Chorio-epithelioma; pseudo Addison's disease; volvulus.
J. J. MOORE—2. Gross surgical pathology.

Friday

A. F. HENNING—9. General surgical clinic.
GEORGE M. LUCAS—10. Gynecological surgery.
C. C. CLARK—11. General surgical clinic.

CHICAGO MEMORIAL HOSPITAL

Tuesday

ARTHUR H. CONLEY and FRED M. MILLER—9. Orthopedic and industrial injury clinic.
JAMES E. FITZGERALD—2. Obstetrical clinic.
JOHN P. O'NEIL, J. WILLIAM PARKER and DORRIN F. RUDNICK—2. Urological clinic.

Wednesday

CHARLES E. KAHLKE, LAWRENCE L. ISEMAN, and M. L. WEINSTEIN—9. General surgical clinic.
FRANK WRIGHT—9. Colloidal state of the blood in post-operative pneumonia.
GEORGE M. LANDAU—2. Phrenico exeresis and treatment of unilateral tuberculosis.
CASPER M. EPSTEIN—2. Oral and plastic surgery.

Thursday

C. R. G. FORRESTER—9. Fracture clinic.
CHARLES J. DRUECK, SR.—2. Proctology.
HARRY L. MEYERS—2. Gynecological clinic.
WILLIAM L. BROWN—2. Radium clinic.

Friday

PETER S. CLARK, LEO M. ZIMMERMAN, and ROBERT A. MELENDY—9. General surgical clinic.
JULIA C. STRAWN and PAUL M. CLIVER—9. Gynecological clinic.

COLUMBUS HOSPITAL

Tuesday

DANIEL A. ORTH, C. O. LINDSTROM and M. L. HANNAN—9. General surgery.
DANIEL A. ORTH—9. Indications and contra-indications for spinal anæsthesia.
CHANNING BARRETT—9. Gynecological operations.
MINAS JOANNIDES—9. Collapse therapy in pulmonary tuberculosis.
M. J. SEIFERT—10. Surgical treatment of ulcer of the stomach.
MINAS JOANNIDES—2. Surgery of the chest.

Wednesday

CHANNING BARRETT—9. Gynecological clinic.
G. N. BEECHER and M. B. BURNS—9. Emergency surgery in industrial injuries.

Thursday

MINAS JOANNIDES—9. Surgical treatment of abscess of lung.
F. MUELLER and F. MUELLER, JR.—9. Transplantation of bone.
WILLIAM GEHL and T. L. CHENOWETH—9. Urological clinic.
G. N. BEECHER and M. B. BURNS—9. Emergency surgery in industrial injuries.

Friday

DANIEL A. ORTH, C. O. LINDSTROM and M. L. HANNAN—9. General surgery.
M. J. SEIFERT—9. General surgery.

U. S. MARINE HOSPITAL

Wednesday

O. E. NADEAU—9. General surgical clinic.

Friday

O. E. NADEAU—9. General surgical clinic.

WESLEY MEMORIAL HOSPITAL

Monday

R. W. MCNEALY—2. Gall-bladder cholecystectomy; inguinal herniotomy.
E. I. GREENE—2. Hyperthyroidism in children.

Tuesday

P. B. MAGNUSON, W. A. HENDRICKS and H. E. E. BARNARD—9. Synovectomy; arthroplasty of knee and jaw; cholecystectomy.
C. B. REED and G. C. RICHARDSON—2. Obstetrical clinic, moving picture demonstration of breech delivery, perineorrhaphy and forceps delivery; demonstration of external measurements of intra-uterine child.
W. B. SERBIN—2. Cæsarean section.

Wednesday

GEORGE H. GARDNER—9. Classic repair of cystocele.
R. W. MCNEALY—11. Breast amputation; gastro-enterostomy.
PHILIP H. KREUSCHER—11. Joint surgery.

Thursday

P. B. MAGNUSON, W. A. HENDRICKS and H. E. E. BARNARD—9. Bone graft of lower spine; bone graft of sacro-iliac joint; extracapsular fusion of hip; cholecystectomy.
GUY VAN ALSTYNE—10. Osteitis tuberculosa multiplex cystica (Juengling).
VICTOR D. LESPINASSE—11. Urological clinic.
M. T. GOLDSTINE—2·30. Gynecological clinic.

Friday

R. W. MCNEALY—9. Ventral and inguinal herniotomy.
EUGENE PERRY—11. Urological surgery.

MUNICIPAL TUBERCULOSIS SANITARIUM

Tuesday

CLEMENT L. MARTIN—9. Perianal tuberculosis.
MINAS JOANNIDES—9. Thoracoplasty; phrenic neurectomy.
HENRY C. SWEANY—11. Pathological conference, demonstration of pathological specimens.

Wednesday

DORRIN F. RUDNICK—9. Nephrectomy for tuberculosis of kidney; operative surgery for tuberculosis of the genito-urinary tract.
FRANK FREMMEL and FRANK SMEJKAL—10. Artificial pneumothorax.
FREDERICK TICE, ALLAN J. HRUBY and K. J. HENRICHSEN—2. Diagnostic clinic.

Thursday

MINAS JOANNIDES and RICHARD DAVIDSON—9. Thoracoplasty; pneumolysis; phrenic neurectomy.
K. J. HENRICHSEN—9. Artificial pneumothorax.

Friday

ALLAN J. HRUBY and K. J. HENRICHSEN—9. Surgical conference.

OUTPATIENT PNEUMOTHORAX CLINIC

2049 Washington Boulevard

MINAS JOANNIDES, E. L. QUINN, EMIL BUNTA, CLARA JACOBSON and GEORGE TSOULOS—9 and 2, daily. Artificial pneumothorax on ambulatory patients.

WOMEN AND CHILDREN'S HOSPITAL

Monday

FRANCES FORD—2. X-ray therapy in malignancies.

Tuesday

BERTHA VAN HOOSEN—9. Gynecological operations.
JOSEPHINE MCCOLLUM and BERTHA VAN HOOSEN—10. Demonstrations of morphine and scopolamine anæsthesia in surgery.
O. ZELEZNY—11. Demonstrations of electrocoagulation therapy.

Wednesday

PEARLE STETLER—9. General surgical operations.
WALBURGA KACIN and CLARA OCHS—2. Obstetrical cases, management under scopolamine anæsthesia.
FRANCES FORD—2. X-ray demonstrations.
PEARLE STETLER—2. Surgical diagnosis of appendicitis in children.

Thursday

ALICE CONKLIN—9. General surgery.
Staff—9. Fracture cases.
MARIE ORTMAYER—10. Urological clinic.
AMELIA GIRYOTAS—11. Carcinoma of the pelvis.
ELOISE PARSONS—2. Endocrine therapy in gynecology, sterility operations.

Friday

MARY E. WILLIAMS—9. Gynecological operations.
CONSTANCE O'BRITIS—11. General surgical operations.
MARY SPIVACK and FLORENCE HARK—2. Obstetrics.
CHARLES FORD—2. X-ray and diathermy therapy.

RESEARCH AND EDUCATIONAL HOSPITAL

Monday

H. B. THOMAS—1. Orthopedic surgery.

Tuesday

CARL A. HEDBLOM and WILLARD VAN HAZEL—9. Thoracic and general surgery.
L. S. SCHULTZ—9. Oral surgery.

Wednesday

ERIC OLDBERG—9. Neurological surgery.
R. B. MALCOLM—9. Neurological surgery.
H. B. THOMAS—1. Orthopedic surgery.
F. H. FALLS—2. Obstetrical and gynecological clinic.

Thursday

CHARLES B. PUESTOW—9. General surgery.
C. M. MCKENNA—10. Urological clinic; cystoscopies.
WILLARD VAN HAZEL—2. Thoracic surgery.

Friday

CARL A. HEDBLOM and WILLARD VAN HAZEL—9. Thoracic and general surgery.
F. H. FALLS—2. Obstetrical and gynecological clinic.

WASHINGTON BOULEVARD HOSPITAL

Tuesday

PAUL C. FOX—9. Gynecological clinic.

Wednesday

A. R. METZ—9. General surgical clinic, presentation of unusual fractures.

Thursday

V. J. O'CONOR—9. Hydronephrosis, etiology and treatment, case reports, X-rays and operative results; suprapubic prostatectomy and transurethral resection of prostate comparatively, indications and results.

CHICAGO LYING-IN HOSPITAL

Staff: FRED L. ADAIR, J. B. DELEE, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, FRANK E. WHITACRE, MANUEL SPIEGEL and H. C. HESSELTINE.

Monday

Staff—2. Obstetrical operations, motion picture demonstration.

Tuesday

Staff—9. Obstetrical and gynecological operations.

Wednesday

Staff—9. Obstetrical and gynecological operations.
Staff—2. Obstetrical clinic, motion picture demonstration.

Thursday

Staff—9. Obstetrical and gynecological operations.
Staff—2. Obstetrical and gynecological dry clinic, motion picture demonstration.

Friday

Staff—9. Obstetrical and gynecological operations.
Staff—2. Obstetrical and gynecological dry clinic, motion picture demonstration.

AMERICAN HOSPITAL

Tuesday

R. B. MALCOLM—9. Surgical clinic, tumors of the neck.
MAX THOREK and PHILIP THOREK—9. Surgical clinic, carcinoma of the rectum.
W. B. GERHARD—9. General surgical clinic.
FRANK E. SIMPSON—2. Radium treatment of carcinoma of the mouth and tongue.
SOLOMON GREENSPAHN and FREDERICK BOWE—2. Management of placenta prævia.

Wednesday

MAX THOREK and PHILIP THOREK—9. Surgical clinic.
HORACE E. TURNER and S. GREENSPAHN—9. Casualty surgical clinic.
DAVIS H. PARDOLL and LEON BEILIN—9. Urological clinic.
FRANK E. SIMPSON—2. Radiological clinic, carcinoma of the breast and female genitalia.

Thursday

BENJAMIN GOLDBERG and JOHN F. PICK—9. Indications and technique for surgery of the chest.
FRANK E. SIMPSON—2. Radiological clinic, indications and contra-indications to radium treatment.

ILLINOIS CENTRAL HOSPITAL

Tuesday

HUGH N. MACKECHNIE—9. General surgery.
PHILIP H. KREUSCHER—9. Orthopedics.

Wednesday

CHARLES PFEIFFER—9. General surgery.
BEVERIDGE MOORE—9. Orthopedics.

Thursday

S. CLEMENT HOGAN—9. General surgery.
VICTOR LESPINASSE—9. Genito-urinary surgery.

Friday

WILLIAM T. HARSHA—9. General surgery.
JAMES GILL—9. Neurologic surgery.
JOHN J. GILL—9. Obstetrics.
CHESTER GUY and A. H. BAUGHER—9. Pathological conference.

HOSPITAL OF ST. ANTHONY DE PADUA

Monday

THOMAS DWYER—2. Demonstrations in surgical pathology.

Tuesday

LAWRENCE RYAN—9. General surgery.
J. J. SPRAFKA—9. General surgery.
O. J. JIRSA—9. Urology.
L. S. TICHY—2. X-ray demonstration.

Wednesday

R. C. CUPLER—9. General surgery.
JOSEPH ZABOKRTSKY—9. General surgery.
F. W. SLOBE—2. Fracture clinic.
M. A. WEISSKOPF—2. Obstetrics.

Thursday

FRANK J. JIRKA—9. Abdominal operations.
F. B. OLENTINE and R. C. DRURY—9. Thyroid surgery and general surgical clinic.
O. J. JIRSA—9. Urology.
L. S. TICHY—2. X-ray demonstration.

Friday

S. E. DONLON—9. General surgery.
A. A. BONA—9. General surgery.
M. A. WEISSKOPF—9. Obstetrics.
F. J. E. EHRMANN—9. General surgery.
WILLIAM BRADLEY—9. General surgery.

GRANT HOSPITAL

Tuesday

ANDRE L. STAPLER—9. General surgery.
F. H. FALLS—9. Gynecology.
E. FISCHMANN—9. Vaginal hysterectomy.
A. G. FREY—9. General surgery.
GEORGE ABELIO—9. General surgery.
E. HESS—10. Urology.

Wednesday

E. SEIDLER—9. Midtarsal resection.
A. G. ZIMMERMAN—9. General surgery.

Thursday

B. H. ORNDOFF—9. Electrosurgery.
W. A. STUHR—9. General surgery.
ANDRE L. STAPLER—2. General surgery.

Friday

SYLVAN COOMBS—9. General surgery.
E. W. FISCHMANN—9. Pus tubes.
A. G. ZIMMERMAN—9. General surgery.

EDGEWATER HOSPITAL

Wednesday—2

N. LOWRY. Electrosurgery in the treatment of carcinoma of the rectum.
LOREN WILDER. Bone transplants.
I. PILOT. Allergic manifestations in arthritis.
R. ELMER. Biliary surgery.
M. A. BERNSTEIN. Bone sarcoma.
M. MAZEL. Traumatic rupture of the spleen.
N. ZEITLIN. Intestinal obstruction.
M. KERN and LOREN WILDER. Surgical and nonsurgical thyroid.
A. F. LASH. Colposcopy.
D. SCHLAPIK. Prostatic resection.
T. PARSCHE. Fractures of the femur.

HOLY CROSS HOSPITAL

Tuesday

J. FRANCIS RUZIC—9. Gynecological operations; cholecystectomy, high spinal anæsthesia.
E. R. CROWDER—9. Some practical considerations regarding the Graham test.
JOHN F. DYBALSKI—10. Hysterectomy, spinal anæsthesia.
VINCENT TORCZYNSKI—11. Appendectomy.

Wednesday

DONALD MONACO—9. Thyroidectomy; lecture on avertin anæsthesia.
A. R. MCCRADIE—10. Hernia operation.
PAUL LAWLER—11. Low cervical cæsarean section.

Thursday

STEPHEN BIEZIS—9. Gynecological operations.
MICHAEL STRIKOL—10. Cholecystectomy.
F. F. FRAIDER—11. Panhysterectomy.
C. H. MCKENNA—11. Cholecystotomy.

Friday

M. J. BADZMIEROWSKI—9. Thyroidectomy; hysterectomy.
RICHARD ROCHE—10. Herniorrhaphy.
ALEXANDER JAVOIS—11. Appendectomy.

LUTHERAN DEACONESS HOSPITAL

Tuesday

GEORGE H. SCHROEDER, JOHN KOUCKY, H. C. WALLACE and G. H. MAMMEN—9. General surgical clinic.

Wednesday

GEORGE H. SCHROEDER, JOHN KOUCKY, H. C. WALLACE, G. H. MAMMEN, R. G. WILLY and G. O. SOLEM—9. Clinical demonstrations.

Thursday

GEORGE H. SCHROEDER, JOHN KOUCKY, H. C. WALLACE and G. H. MAMMEN—9. General surgical clinic.

Friday

GEORGE H. SCHROEDER, JOHN KOUCKY, H. C. WALLACE, G. H. MAMMEN, R. G. WILLY and G. O. SOLEM—9. Clinical demonstrations.

ST. JOSEPH HOSPITAL

Monday

HUGH MCKENNA—2. Review of traumatic surgery with special reference to fractures.

Tuesday

FRANKLIN B. MCCARTY—9. Surgical anatomy, pathology and surgical treatment of diseases of the gall bladder.
RALPH A. KORDENAT—2. Breast tumors.

Wednesday

HUGH MCKENNA—9. Abdominal surgery, surgery of the large intestine.
WALTER W. VOIGT—9. Puerperal sepsis.
THOMAS J. O'DONOGHUE—2. Obstetrical and gynecological operations.

Thursday

WILLIAM H. G. LOGAN—9. Cleft palate and cleft lip operations.
RALPH C. KORDENAT—2. Gall-bladder surgery.

Friday

L. WADE MARTIN—9. Obstetrical clinic.

AUGUSTANA HOSPITAL

Tuesday

N. M. PERCY and O. E. NADEAU—9. Goiter and general surgical clinic.

Wednesday

A. T. LUNDGREN and EARL GARSIDE—9. General surgery.
J. W. NUZUM—9. General surgical clinic.
R. J. ODEN—9. General surgical clinic.

Thursday

N. M. PERCY and O. E. NADEAU—9. Goiter and general surgical clinic.

Friday

A. T. LUNDGREN and EARL GARSIDE—9. General surgery.
J. W. NUZUM—9. General surgical clinic.
R. J. ODEN—9. General surgical clinic.

EVANSTON HOSPITAL

Tuesday

JAMES T. CASE—9. X-ray diagnosis and therapy.
WILLIAM R. PARKES—9. Thyroid clinic.
MARCUS H. HOBART—9. General surgical clinic.
DWIGHT F. CLARK—2. Recent advances in the treatment of common fractures.
MARCUS H. HOBART—2. Fracture clinic.

Wednesday

WILLIAM C. DANFORTH—9. Gynecological operations.
CHARLES E. GALLOWAY—9. Gynecological operations.
JEROME R. HEAD—9. Thoracic surgery.
FREDERICK CHRISTOPHER—2. Demonstration of surgical cases.
ROBERT C. LONERGAN—2. Demonstration of orthopedic cases.

Thursday

WILLIAM C. DANFORTH—9. Gynecological operations.
JOHN L. PORTER—9. Orthopedic operations.
WILLIAM C. DANFORTH—2. Obstetrical clinic.
CHARLES E. GALLOWAY—2. Schiller test for the early diagnosis of carcinoma of the cervix.

Friday

FREDERICK CHRISTOPHER—9. General surgical clinic.
FRANCIS D. GUNN—9. Demonstration of surgical pathology.
CHARLES E. POPE—9. Proctological clinic.
J. EVERETT SANNER—2. Urological clinic.

OAK PARK HOSPITAL

Tuesday

JOHN W. TOPE—9. General surgery.
GORDON SWANSON—9. Orthopedic clinic.
ARTHUR CONLEY—9. Management of fractures of the femur.

Wednesday

RALPH SULLIVAN—9. General surgical clinic, treatment of peptic ulcer.
CHARLES FOX—9. Gynecological operations.
CARL UTHOFF—9. Operative cystoscopy.

Thursday

LOUIS RIVER—9. General surgery.
ADOLPH KRAFT—9. General surgery.
CARL UTHOFF—9. Genito-urinary operations.

Friday

JOHN W. TOPE—9. General surgery.
MEREDITH MURRAY—9. Gynecological operations.

SOUTH SHORE HOSPITAL

Tuesday

AXEL WERELIUS—9. Gastric surgery.
GEORGE G. O'BRIEN—11. General surgery.
CLARENCE S. DUNER and AXEL WERELIUS—2. Symposium on gastric and duodenal ulcer.

Wednesday

HUGH MACKECHNIE—9. Surgery of the colon.
FRANK G. MURPHY—11. Orthopedic clinic.
H. WILLIAM ELGHAMMER, GUY S. VAN ALSTYNE and PAUL R. CANNON—2. Symposium on intussusception.

Thursday

LOUIS D. SMITH—9. Genito-urinary surgery.
CLARA JACOBSON—2. Lung collapse procedures.
C. C. MAHER—4. Cardiac risk in surgery.

Friday

E. A. LUTTON—9. Gynecological clinic.
ANDREW DAHLBERG and WILLIAM HANRAHAN—11. Operative obstetrics.
H. R. COLVER—2. Industrial surgery.
WALTER FISCHER—3. Foot problems.

ST. BERNARD'S HOSPITAL

Monday

W. G. EPSTEIN—2. General surgery.

Tuesday

W. J. MULHOLLAND—9. General surgery.
H. HOFMANN—9. General surgery.
G. M. CUSHING—2. General surgery.
L. B. DONKLE—2. Genito-urinary surgery.

Wednesday

B. C. CUSHWAY and R. J. MAIER—9. Roentgenological demonstration of anomalies of spine.
J. B. HAEBERLIN—9. General surgery.
W. S. HECTOR—9. General surgery.
J. A. PARKER—2. General surgery.
S. L. GOVERNALE and S. S. MARKIEWICZ—2. Gastro-intestinal operations.

Thursday

J. T. MEYER—9. Thyroid surgery.
F. M. PHIFER—9. Genito-urinary surgery.
W. P. GUNN—9. Gynecological operations.
D. A. VLOEDMAN—2. Gynecological operations.
C. C. GUY—2. Demonstration of unusual specimens.

Friday

A. E. MCCRADIE—9. General surgery.
E. A. RACH and F. J. STUCKER—9. Operative obstetrical problems.

EVANGELICAL DEACONESS HOSPITAL

Tuesday

EDWARD HEACOCK—9. General surgery.

Wednesday

PAUL MORF—9. General surgery.

Thursday

A. J. SCHOENBERG—9. Pelvic surgery.

Friday

JOHN PEARL—9. Abdominal surgery; spinal anæsthesia.

JOHN B. MURPHY HOSPITAL

Monday

JOSEPH KEREKES and R. J. MURPHY—2. Rectal treatment of appendiceal and other pelvic abscesses.

Tuesday

H. E. DAVIS—10. Studies of epiphyseal growth disturbances.

Wednesday

M. J. PURCELL—10. Emergency surgery.
O. H. SCHULZ—10. Observations on treatment of pneumonia.

Thursday

F. O. BOWE—9. Treatment of puerperal infections.
H. R. KENNY and S. J. MARK—10. General surgery.

Friday

A. C. GARVY—10. Diagnosis and treatment of skull fractures.
H. R. KENNY and S. J. MARK—10. Pre-operative treatment in abdominal cases.

GARFIELD PARK COMMUNITY HOSPITAL

Tuesday

Staff—9. Symposium on surgery of the stomach with special reference to peptic ulcer. SAMUEL G. PLICE: Diagnosis and medical management. HAROLD N. WAIT: Diagnosis from roentgen ray standpoint. PAUL G. SCHMIDT: Pathology. JOHN R. HARGER: Surgery.
LESLIE F. MACDIARMID—11. General surgery.

Wednesday

CLAUDE WELDY and JOHN J. PFLOCK—9. Medical and surgical aspects of gall-bladder disease; hysterectomy.

Thursday

JOHN M. BERGER and FRANK CHAUVET—9. Gall-bladder disease with special reference to myocardial changes.

Friday

CLARENCE SAELHOF—9. Diapliasic strains of bacteria from renal lesions; experimental production of lesions with spironenia (spirochæta Pallida).
VINCENT J. O'CONOR—9. Tuberculosis of kidney with review of cases; hydronephrosis, plastic repair of nephropexy.

LITTLE COMPANY OF MARY HOSPITAL

Monday

W. D. STADLE—2. Management of eclamptic patients.

Tuesday

L. L. CHARPIER—9. Management of fractures about the elbow.
J. E. LAIBE—10. Treatment of carcinoma of the bladder.

Wednesday

E. D. HUNTINGTON—9. Gastro-intestinal surgery, complications.

Thursday

L. L. CHARPIER—9. Management of compound fractures.
W. A. MALONE—10. Radium treatment of carcinoma of the cervix.

Friday

A. W. WOODS—9. Gynecological repair operations.
E. D. HUNTINGTON—10. Intestinal obstruction.

POST-GRADUATE HOSPITAL

Monday

B. C. CUSHWAY—2. X-ray diagnosis.

Tuesday

H. SOLOWAY—10. Urological clinic.
EMIL RIES—10. Gynecological operations.
D. SCHLAPIK—2. Intra-urethral prostatectomy, moving picture demonstration.

Wednesday

J. C. BOODEL—10. Rectal operations.
LEO ZIMMERMAN—2. Phlebitis.

Thursday

H. L. MEYERS—10. Gynecological operations.
R. A. LIFVENDAHL—11. Gynecological clinic with colposcopic demonstration.

Friday

EMIL RIES—10. Gynecological operations.

ILLINOIS MASONIC HOSPITAL

Tuesday

E. W. WHITE—9. Prostatic and renal surgery.
O. C. RITCH—9:30. Surgery of the kidney.
CLARENCE SAELHOF—11. Tumors of the testicle.

Wednesday

GILBERT FITZ-PATRICK—9. Obstetrical problems.
CHARLES H. PARKES—9:30. Inguinal hernia; cryptorchidism.
CARL F. STEINHOFF—11. Medical phases of thyroid disease.
HUGH N. MACKECHNIE—11. Surgery of the thyroid.

Friday

J. ROSCOE HARRY—9. Medical phases of gall-bladder disease.
CLIFTON K. TIMMONS—9. Gall-bladder surgery.
JOHN F. DAVIS—9:30. Surgery of the colon.
WALTER R. FISCHER—11. Orthopedic problems of the feet.

WEST SUBURBAN HOSPITAL

Monday

HARRY J. DOOLEY—2. Urological clinic.

Tuesday

WILLIAM J. POTTS—9. The healing of fractures.
OSCAR B. FUNKHOUSER—9. Gall-bladder surgery.
THOMAS I. MOTTER—9. General surgery.
JAMES H. SKILES—9. Gynecological clinic.

Wednesday

JOSEPH L. NORTELL—9. General surgery.
FREDERICK H. FALLS—9. Gynecological clinic.

Thursday

CHARLES E. HUMISTON—9. General surgery.
WARD E. POTTER—9. Thyroid clinic.
LOUIS FAULKNER—9. Interesting obstetrical conditions.
PAUL C. FOX—9. Gynecological clinic.
EUGENE C. PIETTE—9. Pathological demonstration.
HOMER HUMISTON—2. Urological clinic.

Friday

HARRY J. STEWART—9. General surgical clinic.

ST. ANNE'S HOSPITAL

Tuesday

T. E. MEANY—10. Orthopedic clinic.
J. L. KNAPP—11. General surgery.
J. B. HARNEY—2. X-ray demonstration.

Wednesday

G. F. THOMPSON—9. Stomach and intestinal surgery.
J. W. MCCONNELL—10. Gynecology.
J. J. GEARIN—11. General surgery.

Thursday

H. J. DOOLEY—9. Urological clinic.
E. P. VAUGHAN—9. Gall-bladder surgery.
E. P. GRAMER—10. Treatment of head injuries.
J. L. FLEMING—11. Pathological obstetrics.

Friday

B. W. MACK—9. General surgery.
Staff—10. Clinical meeting.
D. F. HAYES—11. General surgery.
L. R. HILL—2. Pathological demonstration.

ALBERT MERRITT BILLINGS HOSPITAL

Staff—9, daily. General surgical operations and clinical demonstrations.
WILLIAM ADAMS. Demonstrations in thoracic surgery.
EDMUND ANDREWS. Gall-bladder surgery.
ALEXANDER BRUNSCHWIG. Management of malignant tumors and experimental bone tumors.
E. L. COMPERE, C. H. HATCHER and DR. KEYES. Operations and demonstrations in orthopedic surgery.
LESTER R. DRAGSTEDT. Surgery of the stomach and colon.
C. B. HUGGINS and H. E. HAYMOND. Operations and demonstrations in genito-urinary surgery.
HILGER P. JENKINS. Abdominal surgery.
D. B. PHEMISTER. Bone surgery, operations and demonstrations.

ALEXIAN BROTHERS HOSPITAL

Tuesday

MALCOLM L. HARRIS, AUGUST ZIMMERMAN, ROBERT FLANNERY and GEORGE L. APFELBACH—9. General surgery.
A. WOCHINSKI and EDWARD WHITE—9. General surgery.

EVANGELICAL HOSPITAL

G. ERMAN JOHNSON. Clinical studies of extra-uterine pregnancy.
PERCY E. HOPKINS. Clinical studies of pancreatitis.
CHARLES PAPIK. Treatment of lower limb fractures by fixed traction.
PAUL GEORGE PAPSDORF—Demonstration of models and photographs showing newer methods of the handling of fractures of the maxilla and mandible.

SHRINERS' HOSPITAL

Tuesday

BEVERIDGE MOORE and HAROLD SOFIELD—9. Orthopedic operations.

Wednesday

BEVERIDGE MOORE—2. Demonstration of plaster technique; club foot clinic.

Thursday

BEVERIDGE MOORE and HAROLD SOFIELD—9. Orthopedic operations.

Friday

BEVERIDGE MOORE and HAROLD SOFIELD—2. Out-patient clinic.

FRANCES E. WILLARD HOSPITAL

Tuesday

ALLEN E. STEWART and MILTON OCHS—9. General surgical clinic.
FREDERICK MUELLER—3. Surgery of bones and joints.

Wednesday

OTIS M. WALTER—9. General surgical clinic.
VAUGHN L. SHEETS—10. Diabetic clinic.

Thursday

JOSEPH F. JAROS—9. Thyroid clinic.

Friday

VICTOR L. SCHRAGER—9. General surgical clinic.

SOUTH CHICAGO COMMUNITY HOSPITAL

Tuesday

M. E. FINSKY—2. Avertin anæsthesia, analysis of 200 cases.
LOUIS D. SMITH—2:30. Tuberculosis of the kidney, presentation of case.

Friday

JOSEPH J. LEBOWITZ—2. Fractures and dislocations of the elbow, presentation of cases treated by open operation.
FRANK G. MURPHY—2:30. Fractures of the upper end of the humerus, presentation of cases.
GEORGE G. O'BRIEN—3. Postoperative evisceration, presentation of case.

HENROTIN HOSPITAL

Tuesday

CHANNING BARRETT—9. Gynecological operations.
F. LEE STONE—9. Some problems in tubal patency.

Wednesday

JOHN A. GRAHAM—11. Open reduction of fractures.

SURGERY OF THE EYE, EAR, NOSE AND THROAT

PRESBYTERIAN HOSPITAL AND RUSH MEDICAL COLLEGE

Monday

D. B. HAYDEN—2. Complications of otitis media without rupture of the tympanic membrane.
E. W. HAGENS—2. Unusual laryngeal and bronchial case.
GEORGE E. SHAMBAUGH, JR. and E. W. HAGENS—2. Operations on the tear sac for dacrocystitis.
MAX JACOBSON—3. Neurological aspects.

Tuesday

ROBERT VON DER HEYDT—3. Slit lamp diagnostic clinic.

Wednesday

VERNON LEECH—3. Glaucoma.

Thursday

BERTHA KLEIN—10. Histopathology of fundus.
T. W. LEWIS—2. Discussion of some difficult problems in the operation for correction of the nasal septum.
L. T. CURRY—2. Demonstration of skiagraphs of the sinuses and mastoids.
R. W. WATKINS—2. Nasal findings in allergic cases.
C. L. DOUGHERTY—2. Diathermy and its application to the treatment of nose and throat conditions.

Friday

W. F. MONCREIFF—10. External diseases of the eye and iridocyclitis.
ELIAS SELINGER—3. Fundus.

ALBERT MERRITT BILLINGS HOSPITAL

Tuesday

E. V. L. BROWN—9. Eye clinic.
J. R. LINDSAY—10:30. Ear, nose and throat clinic.
DEWEY KATZ—2. Eye clinic.

Wednesday

LOUIS BOTHMAN—9. Eye clinic.
T. E. WALSH—10:30. Ear, nose and throat clinic.
JOHN STOUGH—2. Eye clinic.
J. R. LINDSAY and G. H. SCOTT—2. Ear, nose and throat operations.

Thursday

P. C. KRONFELD—9. Eye clinic.
G. H. SCOTT and H. B. PERLMAN—10:30. Ear, nose and throat clinic.
DEWEY KATZ—2. Eye clinic.

Friday

DEWEY KATZ—9. Eye clinic.
J. R. LINDSAY and T. E. WALSH—10:30. Ear, nose and throat clinic.
P. C. KRONFELD—2. Eye clinic.
T. E. WALSH and H. B. PERLMAN—2. Ear, nose and throat operations.

EVANGELICAL HOSPITAL

G. HENRY MUNDT. Technique and interpretation of hearing tests and technique and interpretation of tests of the static labyrinth.

ST. LUKE'S HOSPITAL

Monday

EARL VERNON—2. Ophthalmological clinic.

Tuesday

E. FINDLAY and RICHARD GAMBLE—2. Ophthalmological clinic.
J. T. CAMPBELL, JOHN A. CAVANAUGH, HORACE R. LYONS, E. P. NORCROSS, WALTER H. THEOBALD, SYLVIA A. SCIARETTA, ARTHUR J. COOMBS and CLIFFORD L. DOUGHERTY—2. Otolaryngological clinic.

Wednesday

ALVA SOWERS—2. Ophthalmological clinic.
J. T. CAMPBELL, JOHN A. CAVANAUGH, HORACE R. LYONS, E. P. NORCROSS, WALTER H. THEOBALD, SYLVIA A. SCIARETTA, ARTHUR J. COOMBS and CLIFFORD L. DOUGHERTY—2. Otolaryngological clinic.

Thursday

FRANK BRAWLEY and JAMES W. CLARK—2. Ophthalmological clinic.
J. T. CAMPBELL, JOHN A. CAVANAUGH, HORACE R. LYONS, E. P. NORCROSS, WALTER H. THEOBALD, SYLVIA A. SCIARETTA, ARTHUR J. COOMBS and CLIFFORD L. DOUGHERTY—2. Otolaryngological clinic.

Friday

E. FINDLAY and RICHARD GAMBLE—2. Ophthalmological clinic.

COOK COUNTY HOSPITAL

Monday

EARLE B. FOWLER—2. Ophthalmoscopy.
S. PEARLMAN and N. LESHIN—2. Œsophagoscopy and bronchoscopy; surgery of the neck.

Tuesday

JAMES P. FITZGERALD—2. External diseases of the eye.
I. MUSKAT—2. Clinical and surgical otolaryngology; plastic surgery of face and nose.

Wednesday

L. T. CURRY—9. Otolaryngology, clinical and surgical cases.
WILLIAM F. MONCREIFF—9. Ophthalmic neurology and ophthalmoscopy.

Thursday

SANFORD R. GIFFORD—9. Ophthalmic surgery.
CHARLES F. YERGER—11. External diseases of the eye.
S. PEARLMAN and N. LESHIN—2. Œsophagoscopy and bronchoscopy; surgery of the neck.

Friday

T. C. GALLOWAY and M. T. LAMPERT—10. Malignancy about the head, diathermy.
JAMES P. FITZGERALD—2. Ophthalmic surgery.
I. MUSKAT—2. Clinical and surgical otolaryngology; plastic surgery of face and nose.

CHICAGO EYE, EAR, NOSE AND THROAT HOSPITAL

Tuesday

H. B. FULLER—9. Mastoid surgery.
WILLIAM A. HOFFMAN and WILLIAM LINGARD—9. Eye, ear, nose and throat clinic.
WILLIAM A. FISHER—9. Cataract operations.
L. SAVITT—10. Removal of tonsils by diathermy.
OSCAR B. NUGENT—11. Eye clinic.
O. M. STEFFENSON—11. Ear, nose and throat clinic.
T. S. KAMMERLING—2. Eye, ear, nose and throat clinic.

Wednesday

O. M. STEFFENSON—9. Tonsil dissection.
OSCAR B. NUGENT—9. Cataract operations.
WILLIAM A. HOFFMAN and WILLIAM LINGARD—9. Nasal surgery and eye, ear, nose and throat clinic.
OSCAR B. NUGENT—11. Eye clinic.
O. M. STEFFENSON—11. Ear, nose and throat clinic.
L. SAVITT—11. Ear, nose and throat clinic.
H. B. FULLER—2. Eye, ear, nose and throat clinic.

Thursday

WILLIAM A. FISHER—9. Eye operations.
WILLIAM A. HOFFMAN and WILLIAM LINGARD—9. Eye, ear, nose and throat clinic.
T. S. KAMMERLING—9. Surgery of the nasal accessory sinuses.
L. SAVITT—10. Physical measures in otolaryngology.
O. M. STEFFENSON—11. Ear, nose and throat clinic.
L. SAVITT—11. Ear, nose and throat clinic.
OSCAR B. NUGENT—11. Eye clinic.
T. S. KAMMERLING—2. Eye, ear, nose and throat clinic.

Friday

O. M. STEFFENSON—9. Tonsil dissection.
WILLIAM A. HOFFMAN and WILLIAM LINGARD—9. Eye, ear, nose and throat clinic.
OSCAR B. NUGENT—9. Fundus photography and pathology.
H. B. FULLER—10. Functional testing.
O. M. STEFFENSON—11. Ear, nose and throat clinic.
OSCAR B. NUGENT—11. Eye clinic.
H. B. FULLER—2. Eye, ear, nose and throat clinic.

MICHAEL REESE HOSPITAL

Monday

H. S. GRADLE—2:30. Eye surgery.

Tuesday

S. J. PEARLMAN—9. Bronchoscopic clinic.
M. L. FOLK—2. Eye surgery.

Wednesday

SAMUEL SALINGER—9. Nasal fractures; plastic of the nose.
M. L. FOLK—2. Eye clinic.
H. S. GRADLE—2:30. Surgical eye clinic.
ROBERT VON DER HEYDT—3. Slit-lamp demonstration.

Thursday

CASPER EPSTEIN—9. Cleft palate and harelip.
J. C. BECK and M. REESE GUTTMAN—10. Voice production following laryngectomy.

RESEARCH AND EDUCATIONAL HOSPITAL

Otolaryngological Staff: F. L. LEDERER, W. H. THEOBALD, J. J. THEOBALD, G. S. LIVINGSTON, E. A. BREDLAU, N. FOX, S. L. SHAPIRO, I. G. SPIESMAN, P. A. HALPER, A. C. KANE, A. COOMBS, J. HARNED, O. VAN ALYEA, M. GUTTMAN, S. MORWITZ, M. OSTROM, B. LAMBRAKIS, E. HARTLETT, H. KLAWANS, L. FISHMAN, H. WADSWORTH, J. BELLOWS and N. FABRICANT.
Ophthalmological Staff: HALLARD BEARD, M. L. FOLK, H. J. SMITH, S. WOLF, S. KAUFMAN, CARL APPLE and J. W. CLARK.

Monday

Staff—2. Otolaryngological out-patient clinic.

Tuesday

Staff—9. Ophthalmological clinic, operations and demonstrations.
Staff—10. Otolaryngological out-patient clinic.
Staff—2. Otolaryngological clinic, operations and demonstrations.

Wednesday

Staff—9. Eye clinic.
Staff—10. Otolaryngological out-patient clinic.
Staff—2. Otolaryngological out-patient clinic.
Staff—4. Otolaryngological seminar.

Thursday

Staff—9. Otolaryngological operations.
Staff—9. Eye clinic.
Staff—10. Otolaryngological out-patient clinic.
Staff—2. Otolaryngological clinic, operations and demonstrations.
Staff—2. Otolaryngological out-patient clinic.

Friday

Staff—9. Eye clinic, operations and demonstrations.
Staff—10. Otolaryngological out-patient clinic.
Staff—2. Otolaryngological out-patient clinic.

WESLEY MEMORIAL HOSPITAL

Tuesday

ROBERT BLUE—9. Eye clinic.
OTIS H. MACLAY—10. Nasal sinus surgery and demonstration of culture technique for the examination of maxillary and frontal sinuses.

Wednesday

THOMAS P. O'CONNOR—10. Otolaryngological clinic.
A. H. ANDREWS, E. E. DILLON, A. H. ANDREWS, JR.—2. Mastoid operations on cadaver showing simple, modified and radical operations, with a discussion of the indications for each.

Thursday

CHARLES B. YOUNGER—9. Nose, throat and ear operative clinic.

Friday

ROBERT BLUE—9. Eye clinic.
OTIS H. MACLAY—10. Nose, throat and ear clinic.

ST. BERNARD'S HOSPITAL

Friday

PHILIP O'CONNOR—2. Surgery of the eye, dry clinic.

ILLINOIS EYE AND EAR INFIRMARY

Monday

R. Von der Hyedt—2. Eye surgical clinic.
S. Salinger—2. Ear, nose and throat surgical clinics.

Tuesday

S. J. Meyer—2. Eye surgical clinic.
G. Livingston—2. Ear, nose and throat surgical clinic.

Wednesday

Dwight C. Orcutt—2. Eye surgical clinic.
J. Cavanaugh—2. Ear, nose and throat surgical clinic.

Thursday

E. K. Findlay—2. Eye surgical clinic.
T. J. Novak—2. Ear, nose and throat surgical clinic.

Friday

T. D. Allen—2. Eye surgical clinic.
Alfred Lewy—2. Ear, nose and throat surgical clinic.

MOUNT SINAI HOSPITAL

Monday

J. C. Beck, M. R. Guttman and associates—2. Septum cases of uncommon variety; discussion and presentation of cases of malignancy about the nose and pharynx; carcinoma of the larynx, presentation of laryngectomized patients.

Wednesday

A. Lewy, S. M. Morwitz and associates—2. Sepsis associated with ear disease; cases of labyrinthitis; treatment of atrophic rhinitis.

Friday

James E. Lebensohn—9. Operations for cataract and squint.
J. Lifschutz, M. A. Glatt and associates—2. Otogenic septic meningitis with recovery; otogenic sepsis with death following blood transfusion; tracheobronchial Hodgkin's disease; bronchial melanoma; laryngeal chondroperichondritis.

OAK PARK HOSPITAL

Tuesday

Howard Riordan—9. Demonstration of new nasopharyngoscope on the cadaver and living.

Thursday

Howard Riordan—9. Treatment of maxillary sinusitis with the cold quartz lamp; new method of treatment of maxillary polypi by diathermy.

Friday

Georgiana Theobald—9. Demonstration of eye tumors; ophthalmic surgery.

WASHINGTON BOULEVARD HOSPITAL

Tuesday

L. McBride—2. Nose and throat clinic.

Wednesday

Virgil Westcott—2. Eye clinic.

MERCY HOSPITAL

Tuesday

George T. Jordan—9. Nasal ganglion.
L. G. Hoffman—9. Cataract extractions.
C. H. Christoph—9. Bronchoscopy.

Wednesday

George Musgrave and Alfred Paisley—9. Frontal sinus operation, local anæsthesia; modified radical mastoid operation with complete removal of flap; presentation of cases.

Thursday

Ulysses J. Grim—9. Radical antrum and mastoid.
Deno O'Connor and Ray Kerwin—9. Ocular tumors.
Carl Schaub—9. Focal infection in iritis.

CHILDREN'S MEMORIAL HOSPITAL

Wednesday

George S. Livingston and Max T. Lampert—9. Intracranial complications of otitic origin, review of cases.
Maurice H. Cottle—9. Focal infections of the nose and throat in arthritis; methods of study and treatment.
Glenn J. Greenwood—9. Intranasal surgery in children.
George S. Livingston—9. Introduction of iodized oil into the bronchi in the study of bronchiectasis.
Richard Gamble—2. External eye diseases in children.
Elmer Vorisek—2. Fundus diseases in children.
George P. Guibor—2. Orthoptic training in the treatment of strabismus.

CHICAGO MEMORIAL HOSPITAL

Monday

Richard H. Street and Richard W. Watkins—2. Otolaryngological clinic.

Tuesday

Herman P. Davidson and Glenway W. Nethercut—2. Eye clinic.

Wednesday

Alfred E. Lewy and Irving I. Muskat—2. Otolaryngological clinic.

COLUMBUS HOSPITAL

Monday

Michael Goldenburg—2. Emergency surgery of the eye.

Wednesday

G. B. Lambrakis—9. Indications for operative treatment in acute mastoiditis.
S. Sciaretta—9. Otolaryngological clinic.
Michael Goldenburg—2. Eye surgery.

Friday

Michael Goldenburg—2. Eye surgery.

AUGUSTANA HOSPITAL

Wednesday

Alfred Murray—2. Eye, ear, nose and throat clinic.

ST. JOSEPH'S HOSPITAL

Wednesday

AUSTIN A. HAYDEN—9. Otolaryngological clinic: Tonsillectomy hæmostatis, relative importance of blood clotting and bleeding time; stumps; atresia of the posterior choanal orifices, demonstrated by wax models. Hearing conservation, functional tests; mastoid surgery. Submucous resection; sinus surgery; treatment of fractures. Operations and demonstration of cases supplemented by lantern slides, motion pictures, etc.

ST. MARY OF NAZARETH HOSPITAL

Tuesday

GEORGE W. MAHONEY—9. Eye clinic.
J. J. KILLEEN—9. Ear, nose and throat clinic.

Thursday

J. J. KILLEEN—9. Ear, nose and throat clinic.

Friday

E. ROLING—9. Eye clinic.

WOMEN AND CHILDREN'S HOSPITAL

Tuesday

ALICE K. HALL—10. Nose and throat clinic.

Wednesday

FRANCES HAINES—10. Nose and throat clinic.

GRANT HOSPITAL

Wednesday

S. H. SOBOROFF—9. Ear, nose and throat clinic.
GEORGE DENNIS—9. Eye, ear, nose and throat clinic.

SOUTH CHICAGO COMMUNITY HOSPITAL

Tuesday

GEORGE E. PARK—3. The center of ocular rotation in the horizontal plane.

FRANCES E. WILLARD HOSPITAL

Thursday

WILLARD D. BRODE—10. Surgery of throat and nose.

LITTLE COMPANY OF MARY HOSPITAL

Wednesday

H. T. NASH—10. Emergency surgery of the eye.

PASSAVANT MEMORIAL HOSPITAL

Friday

J. GORDON WILSON, JOHN DELPH, CARL BOOKWALTER and ELLISON ROSS—9. Ear, nose and throat clinic.
SANFORD GIFFORD, WILLIAM MANN, JR. and RALPH DAVIS—11. Ophthalmology.

WEST SIDE HOSPITAL

Monday

JAMES CLARK—2. Ear, nose and throat clinic.

Tuesday

A. E. LUNN—2. Eye, nose and throat clinic.

Wednesday

A. E. LUNN—2. Eye, nose and throat clinic.

Thursday

JAMES CLARK—2. Ear, nose and throat clinic.

Friday

A. E. LUNN—2. Eye, nose and throat clinic.

WEST SUBURBAN HOSPITAL

Monday

ROBERT H. GOOD—2. Surgery of the nose, motion picture demonstration.

Tuesday

JOHN J. THEOBALD—2. Mastoid surgery.

Wednesday

GEORGIANA THEOBALD—2. Eye pathological exhibit.

SOUTH SHORE HOSPITAL

Monday

JOHN W. STANTON—2. Mastoiditis and its complications.

Thursday

JOHN W. STANTON—11. Otolaryngological surgery.

AMERICAN HOSPITAL

Tuesday

HARRY L. POLLOCK and ASSOCIATES—2. Ear, nose and throat clinic.

Wednesday

OSCAR KRAFT—2. Ophthalmological clinic.

ILLINOIS MASONIC HOSPITAL

Tuesday

M. H. COTTLE—10. Some advances in mastoid work.
B. M. WOLIN—10. Tonsil surgery in the poor risk cases.
H. E. TAYLOR—10. Conservative surgery of the nose.

JACKSON PARK HOSPITAL

Tuesday

H. E. L. TIMM—1. Timm's modification of Sluder tonsillectomy.

ILLINOIS CENTRAL HOSPITAL

Tuesday

HIRAM SMITH—9. Eye clinic.

Wednesday

JAMES H. MCLAUGHLIN—9. Nose and throat surgery.

EVANSTON HOSPITAL

Tuesday

THOMAS C. GALLOWAY—9. Otolaryngological clinic.

Thursday

HOWARD C. BALLENGER—9. Otolaryngological clinic.

Friday

GAIL R. SOPER—2. Lesions of the fundus oculi, lantern slide demonstration.

RAVENSWOOD HOSPITAL

Wednesday

A. N. MURRAY—10:30. Malignancies of the eye.

MOTHER CABRINI HOSPITAL

Tuesday

NOAH FOX, J. W. HARNED, JR. and F. M. CRAGE—2. Ear, nose and throat clinic.

Friday

NOAH FOX, J. W. HARNED, JR. and F. M. CRAGE—2. External ethmo-frontal operation.

GARFIELD PARK HOSPITAL

Wednesday

ROBERT H. GOOD—2. Submucous resection of the septum on the cadaver and living, motion picture demonstration and discussion.

Friday

ROBERT H. GOOD—2. Intranasal tear sac operation on the cadaver and living, motion picture demonstration and discussion.

ST. ANNE'S HOSPITAL

Tuesday

B. T. GORDON—9. Nose and throat clinic.

Wednesday

W. K. GRAY—9. Eye and ear clinic.

JOHN B. MURPHY HOSPITAL

Monday

E. F. GARRAGHAN—2. Eye operations.

Tuesday

L. H. WOLF and PAUL WOLF—10. Mastoid surgery.

Friday

GEORGE W. MAHONEY—9. Cataracts.